Anæsthesia

JOURNAL OF THE ASSOCIATION OF ANÆSTHETISTS
OF GREAT BRITAIN & IRELAND.

VOLUME 15 · JANUARY 1960 · NUMBER 1

The advantages of 'Fluothane' in tonsillectomy

'FLUOTHANE' offers important advantages when used for tonsillectomies, especially in children.

Induction is rapid and easy and there is quick relaxation of the jaw and pharynx which facilitates intubation and access by the surgeon.

The amount of bleeding is reduced and a notable feature is the patient's rapid recovery with little or no vomiting.

Being non-inflammable, 'Fluothane' can be used with safety in procedures involving diathermy or electrocautery.

Incidentally . . .

"Chlorhexidine is regarded as the most suitable disinfectant for anaesthetic equipment."

Brit. J. Anaes., 1959, *31*, 363

Chlorhexidine is an I.C.I invention presented under the trade name 'Hibitane'.

Fluothane—*an I.C.I. discovery*

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1960

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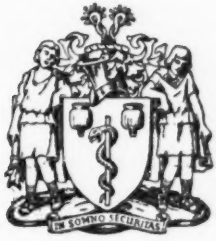

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CONTENTS

	Page
EDITORIAL	1
ANÆSTHESIA IN OPERATIONS FOR THE RELIEF OF PORTAL HYPERTENSION — Ronald A. Bowen	3
HALOTHANE — Denis ApIvor	11
HALOTHANE AND HYPOTENSION — G. E. Hale Enderby	25
POST-OPERATIVE POSTURAL HYPOTENSION — W. Sniper	33
TOTAL BRONCHOSPASM AND LUNG MASSAGE — V. P. Smolnikoff	40
FUNCTIONAL INFUNDIBULAR STENOSIS — H. A. Condon *and* P. F. S. Lee	45
DRUGS WHICH STIMULATE AFFECTIVE BEHAVIOUR – PART I (*Part II will appear in our next issue*) — Allen B. Dobkin *and* John H. Harland	48
EFFECT OF DECAMETHONIUM UPON CONDITIONED REFLEXES IN RATS — Stanley A. Feldman	55
THE RESISTANCE OF EXPIRATORY VALVES IN ACTUAL USE — A. R. Hunter	61
SUMMARIES OF PAPERS READ AT THE SCIENTIFIC SESSION	
Ventilation nomograms during anæsthesia — J. F. Nunn	65
Changes in respiratory physiology of significance to anæsthesiologists — M. T. Jenkins	66
Ventilation during anæsthesia — R. Woolmer	66
The early history of ether — F. Cartwright	67
Is it safe to employ hypotensive anæsthesia in the elderly ? — W. N. Rollason *and* J. M. Hough	69
Anæsthetic methods in animals — Barbara Weaver	70
Induction of anæsthesia in children — R. J. Hamer Hodges	71
Some pain threshold studies with particular reference to thiopentone — J. Clutton-Brock	71
The experimental and clinical use of deep hypothermia — D. Benazon	72
The full stomach in anæsthesia for trauma — L. Wolfson	73
NEW INVENTIONS	
Oral endotracheal connection — G. H. Caiger *and* D. A. S. Sichel	75
A two-way union for double lumen tubes — G. M. J. White	77
Automatic unidirectional flow valve — D. F. Rees	79
Perspex endotracheal adapter — H. H. Samson	81
ANÆSTHETIC LITERATURE	84
CORRESPONDENCE	
Spinal analgesia and unrelated paraplegia — F. N. Prior	90
Bronchospasm — Margaret C. Watkinson	90
Collapse after halothane — H. R. Marrett	91
Anæsthesia for Fredet-Rammstedt's operation — S. H. S. Love *and* G. W. Black	92
OBITUARY	
George F. Rawdon Smith	93
BOOK REVIEWS	
Automatic ventilation of the lungs — W. W. Mushin, L. Rendell-Baker *and* P. W. Thompson	94
Anesthesia for infants and children — Robert M. Smith	94
Resuscitation of the unconscious victim — Peter Safar *and* Martin C. McMahon	95
Principles and practice of obstetric anæsthesia — J. Selwyn Crawford	95
ASSOCIATION NEWS	97
FACULTY NEWS	101
COMMONWEALTH AND FOREIGN NEWS	101

ASSOCIATION OF ANÆSTHETISTS OF GREAT BRITAIN AND IRELAND

NEWSLETTER

JANUARY 1960

The Annual Meeting at Stratford-upon-Avon was attended by about three hundred members and visitors. This is the largest "congress" which the town has had to accommodate, and the numbers wishing to come to the Dinner made some overcrowding unavoidable. In spite of this the Meeting was very successful, and it is felt that the policy of varying the place of meeting is generally popular.

In September 1960 the Second World Congress of Anæsthesiologists is being held in Toronto, and national Meetings, by agreement, are being held on a rather smaller scale. The Association will, therefore, meet in early November at The Royal College of Surgeons of England. In 1961, however, a full-scale Meeting is to be held in Dublin.

Council is elected annually by the Annual General Meeting and usually, but not invariably, those nominated by Council are elected. Four or five members retire annually and Council tries by its nominations to get the help of individuals from all parts of Great Britain and Ireland, places with special interests tending to have generous representation. There are at present two members from Scotland, one from Ulster and one from Eire and a balance is maintained between members from the provinces and from the London area.

A statement in the January issue of *Anæsthesia* gives an indication of the amount of work and thought which has been devoted to Journal affairs by a special Sub-Committee, the Editorial Board and by Council itself.

The safety of anæsthesia for surgery in the dental chair has also been considered and a special Sub-Committee has been appointed to report to Council. The help of the Faculty of Anæsthetists has been asked to determine the amount of instruction available at the different centres to students and post-graduates. Practical instruction is limited by the number of patients available at the dental hospitals and by the large number of dental as well as medical students. The possibility of organising a one- or two-day course of practical instruction, making use of closed circuit television is being considered. The Association has decided to offer a Prize of two hundred guineas for an essay which it is hoped will help to stimulate interest in the problem of dental anæsthesia. Details will be found in the January issue of the Journal. Council has presented evidence to the Joint Working Party on Medical Staffing Structure in the Hospital Service in a memorandum which has been circulated to members. Other activities have included the preparation and giving of evidence before the Interdepartmental Committee on Drug Addiction. From the Anæsthetists Group Committee of the British Medical Association a request for the payment of a fee to anæsthetists for the use of their own apparatus for domiciliary anæsthesia has been forwarded to Committee B of the Medical Whitley Council.

The increase in the new Research and Education Trust Fund is most satisfactory, but that this is almost entirely due to the single handed efforts of the President is something of a reproach to the rest of us.

The Association's subscription, which has remained unchanged since 1932, has at last had to be raised to meet the increasing costs of running the Association. During 1960 members are asked to make an especial effort to get new members. Of the organisations for anæsthetists it is the Association which is most actively concerned with working conditions, organisation and staff problems. The next few years are going to be critical and it is most important that the Association be strong.

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Hon. Secretary

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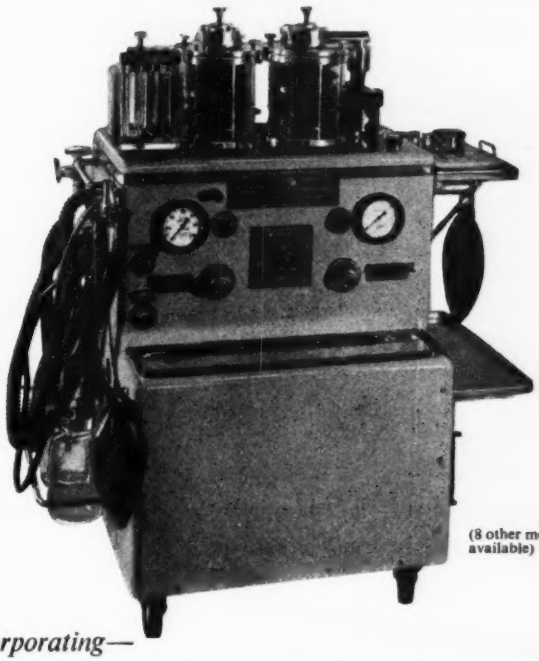

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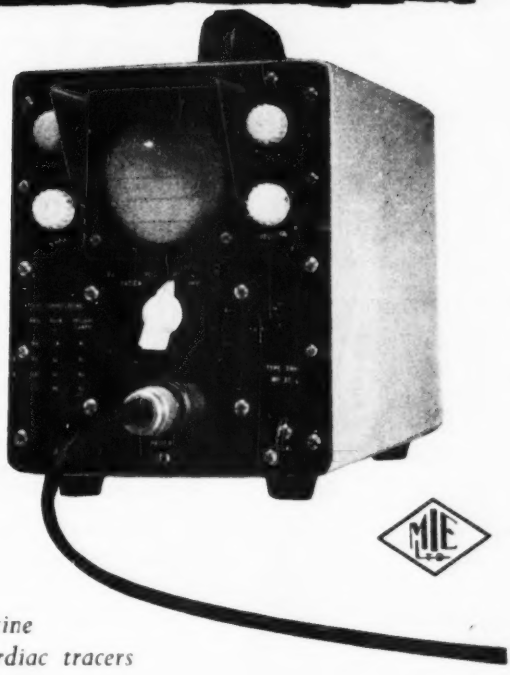

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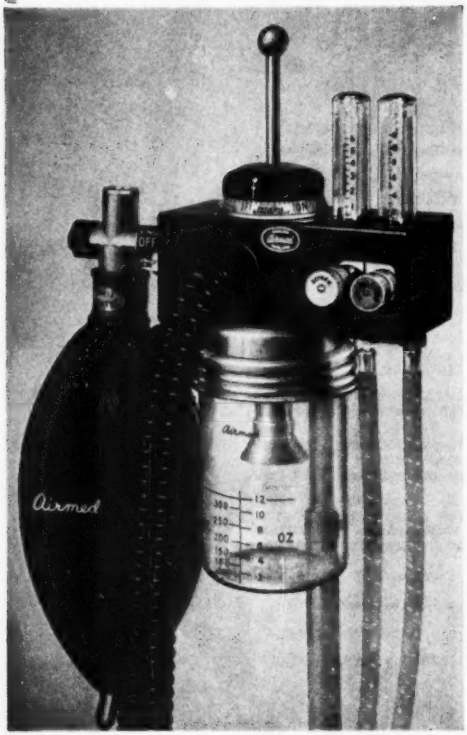

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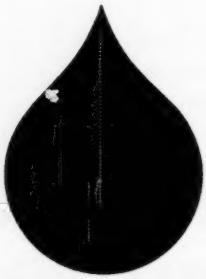

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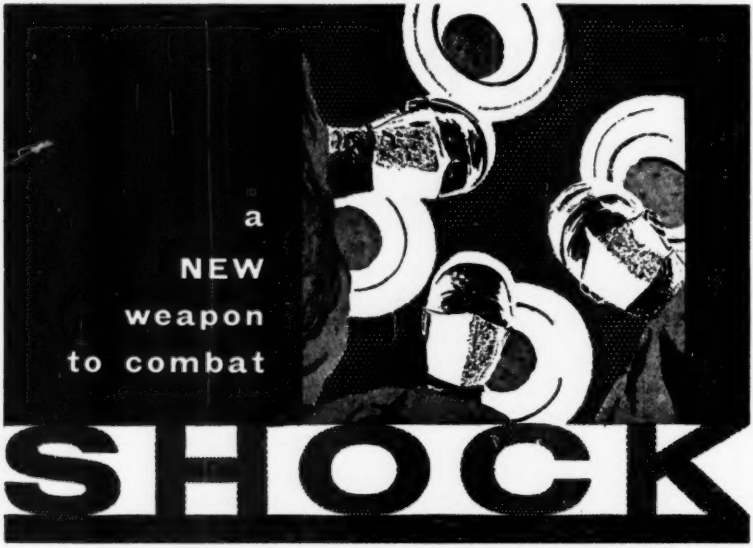

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EDITORIAL

Many readers will have heard more or less inaccurate rumours of an amalgamation between *Anæsthesia* and our contemporary the *British Journal of Anæsthesia.* In order that the facts may be known, it was thought desirable to publish the following agreed statement simultaneously in the two journals.

At the Annual Meeting of the Association of Anæsthetists held in Southport in 1958, the Honorary Treasurer had to present a balance sheet, which, for the first time, showed a considerable excess of expenditure over income. This was to a large extent due to a fall ir the income received from our journal. It was appreciated that this was mainly attributable to the fact that a larger than usual number of subscriptions to the Journal were outstanding at the time of closing the year's accounts. Nevertheless, Council was anxious about the situation and set up a sub-committee to look into the matter. This sub-committee confirmed that it was likely that this loss would be offset to some extent by a considerable profit this year—(a forecast which has been proved correct). However, the sub-committee had wide terms of reference and felt that something would be gained by examining the whole position of journals of anæsthesia in this country. It realised that there was much to be said for one journal representative of the best work and it decided to explore with the Board and Management of the *British Journal of Anæsthesia* the possibility and desirability of a merger. The Board of Management and the Publishers of the *British Journal of Anæsthesia* showed that they were willing to co-operate and suggested conditions on which an

amalgamation of the two journals might be achieved. Although many in the Council of the Association saw the advantages of one journal, there were misgivings on the effect of the greatly increased subscription which would become necessary, and there was doubt as to the practicability of the proposed editorial arrangement. Finally, there was a minority who considered that it was desirable to have two journals of anæsthesia and that competition was a good thing.

The Council considered that in the absence of unanimous enthusiasm for the move it would be undesirable to proceed further.

It is hoped that both these journals will continue to go from strength to strength and remain worthy of the high standard of the specialty in this country.

Anæsthesia in operations for the relief of portal hypertension

RONALD A. BOWEN, MB, BS, FFARCS

Consultant Anæsthetist
St Bartholomew's Hospital, London.

The pressure in the portal vein in health varies between about 40mm and 210mm water[1]. Raised pressure in the system may result from obstruction to the vein as its branches ramify in the liver, as in cirrhosis, or from extra-hepatic causes. These include congenital portal vein obliteration, thrombosis from trauma or infection, compression or invasion by abdominal neoplasm, right heart failure and constrictive pericarditis. The net effect of all these conditions is the same, namely slowing or reversal of flow in the portal system and a natural attempt at by-pass and reduction of pressure by increased flow through the portal-systemic anastomoses. These are numerous, but the most important are those in the lower œsophagus and upper stomach. Varices developing in this situation may erode and rupture, with severe and sometimes fatal hæmatemesis. Such bleeding and melæna apart, presenting symptoms and signs may be anæmia, splenomegaly, ascites, jaundice or stupor.

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Although not curative, operation may prolong life by reducing the risk of hæmorrhage. The most effective is porta-caval anastomosis. In this, the portal vein in mobilised and divided, the hepatic end tied off and the distal end joined end-to-side to the inferior vena cava. Access to these veins is difficult due to their depth in the abdomen and the presence of very vascular connections in the neighbourhood may make it dangerous to proceed.

An alternative method of decompression is lieno-renal anastomosis in which, after splenectomy, the splenic vein is anastomosed to the left renal vein. This is not so effective an operation, as the stoma is smaller.

Other forms of portal-systemic anastomosis, such as inferior mesenteric-caval shunt may cause some reduction in pressure.

Proximal gastric transection consists in isolating and ligating the

vascular connections to the œsophagus and stomach. The latter is then transected close to the cardia and all varices tied off. The cut edges of the stomach are finally approximated by suture.

In proximal gastric resection, in addition to the ligation of external vascular connections the portion of stomach containing varices between cardia and incisura angularis is removed.

Hepatic arterial ligature (Rienhoff's operation) may be tried in an attempt to reduce hepatic congestion.

Oesophagotomy and ligation of varices, a temporary expedient to arrest hæmorrhage involves incision of the œsophagus and obliteration of the columns of veins by running suture.

Small varices may be thrombosed by the injection of sclerosing solutions.

SPECIAL CONSIDERATIONS

Duration of operation

In this series the average operating time has been 180 minutes. A long-acting relaxant is therefore preferable and tubocurarine has always been employed. A Blease Pulmoflator has been used in all the later cases to maintain controlled respiration. Such a machine is a boon, since it is constant in performance and leaves the anæsthetist free to chart records without anxiety.

Hepatic function

The assessment of liver function is notoriously difficult, but two estimations give some helpful information, those of the serum albumin and pseudocholinesterase, both substances being formed in the liver. Hunt considers that operation is contra-indicated where the albumin is less than 3.2g/100ml[2]. The pseudocholinesterase level is of particular interest to the anæsthetist in relation to the duration of scoline apnœa and will be considered later.

Dundee[3], investigating the effect of barbiturates on the liver by measuring the post-operative excretion of urinary urobilinogen, found that liver dysfunction occurred in an appreciable number of patients who received doses of thiopentone exceeding 750mg. This amount has been exceeded on many occasions in patients in this series. In thirty-four mild cirrhotics, for example, doses as large as 1000mg have produced no clinical signs of liver damage. However, three patients developed transient jaundice or stupor following dosage of 400mg or less.

In seventy-five advanced cirrhotics, sixty-two patients received doses up to 800mg without clinical signs of liver damage. Six patients developed transient jaundice or coma following doses of 800mg or less.

Seven patients died in liver failure after thiopentone dosage of 600mg or less.

In practice the sleep dose may most accurately be assessed by giving no more than 100mg thiopentone at a time. Each increment is injected rapidly, pausing for about fifteen seconds between injections. When the eyelash reflex is absent, a further 100mg thiopentone is given before injecting scoline.

Hæmorrhage

Bleeding during these operations is considerable due to the raised venous pressure and to numerous natural portal-systemic anastomoses which may be very friable.

A reliable transfusion should therefore be set up in the anæsthetic room after induction. A Frankis Evans needle is recommended, as it permits cannulation without cutting down and the risk of vein damage during limb movement is negligible.

ANÆSTHETIC TECHNIQUE

This has been standardised for several years. Premedication is by pethidine 100mg with atropine 0.65mg. Sedation following this combination is poor, but it is less likely to cause liver damage than papaveretum-scopolamine. Induction is by thiopentone followed by scoline through the same needle. The lungs are then inflated with oxygen. Laryngoscopy is followed by thorough spraying of the trachea with 6–7ml of 4 per cent lignocaine and intubation with a cuffed Magill tube. The lungs are then inflated with nitrous oxide-oxygen and trichlorethylene in semi-closed circuit until spontaneous respiration returns. In small children a nitrous oxide–oxygen and trichlorethylene induction precedes the injection of a small dose of thiopentone, scoline again being used to aid intubation. A plain Magill tube and pack are preferred. An intravenous infusion is then set up in the left arm, the sphygmomanometer cuff being applied to the right. Immediately before entry into the theatre a test dose of tubocurarine 20mg is injected intravenously. The patient is placed on the table in the 'dorsal hornpipe' position[4]. In this, the arms are folded across one another so that the hands lie over the opposite shoulders. A long piece of elastoplast is fastened to the underside of the operating table on the left side and stuck along the arms before being securely attached beneath the right side of the table. In this way both arms are elevated from the chest, allowing free respiration and easy access to the transfusion apparatus. The only difficulty experienced with this position is that the auscultatory method of recording blood pressure is unreliable, and palpation is now used. A Waters' canister is secured

to the headpiece of the table and connected to the 'Pulmoflator'. A gas flow of nitrous oxide 4 litres per minute and oxygen 2 litres per minute is used, but if ascites or other event embarrasses breathing the oxygen percentage is increased.

It is important before starting the patient on a mechanical respirator to ensure that spontaneous breathing is absent or minimal, so if the dose of tubocurarine already given has not produced these conditions a further 5–10mg are injected. The machine is then turned on.

Special investigations are carried out during operation which demand apnœa. These are per-cutaneous splenic venography and after the abdomen has been opened, estimation of portal venous pressure and perhaps portal venography. The first of these may require up to thirty seconds for its performance and in these circumstances it is preferred to turn off the Pulmoflator and resort to vigorous hyperventilation by hand in advance.

A close watch is kept on blood-loss from observation of the swabs and suction bottle, not forgetting the overhanging towels and floor. The danger of underestimating hæmorrhage is considerable. The majority of patients received 2 litres (four bottles) of blood during operation.

In anastomotic procedures a careful check is kept on the adequacy of curarisation, since hiccough or straining during the suture of delicate vessels must be especially avoided. The anastomosis per se in porta-caval shunt may occupy forty minutes or more. In the latter operation the pleura may be accidentally opened. In spleno-renal anastomosis it is opened by intent. The anæsthetist should therefore be satisfied that the lungs are fully inflated before the chest is closed. This is best done by hand. A second venous pressure measurement is made after completion of the anastomosis.

Reversal of the relaxant is started during skin closure. Neostigmine 1.25mg. mixed with atropine 0.65mg is injected. If the response is not adequate another mixed dose in the same proportions is given. The cuff and harness of the endotracheal assembly are then released and thorough oro-bronchial toilet carried out. For tracheo-bronchial suction a small-size rectal tube, well lubricated, is preferred as being the least traumatic and of ample length.

In the majority of patients an oral airway is not tolerated and awakening in the theatre or recovery room is common. It is essential at this point to provide intravenous sedation, as it may be some time before drugs given subcutaneously or intramuscularly by the nursing staff in the ward take effect. Pethidine 20–30mg is therefore given in the theatre to most patients. It rapidly produces analgesia without loss of protective reflexes and the first dose required in the ward may be delayed for two hours or more.

DIFFICULTIES DURING ANÆSTHESIA

Scoline apnæa

After use for some years, tubo-curarine was abandoned as an aid to intubation in favour of scoline. This choice may seem unwarranted in view of the possibility of low pseudocholinesterase levels in patients suffering from liver disease. However, the relaxation and especially the tracheal quiescence produced by scoline are superior. The trachea is always sprayed with analgesic solution, as this permits toleration of the endotracheal tube at the lightest levels of anæsthesia.

Lehmann[5] considers the critical level of the serum pseudocholinesterase to be 25 units and limited experience bears this out. Of sixty-nine patients given scoline prior to intubation, one case of prolonged apnœa occurred. To this patient, having a pseudocholinesterase level of 13 units, 45mg scoline were inadvertently administered, causing an apnœa of thirty minutes. In eight of the remaining patients the enzyme level was below the normal lower limit of 55 units and ranged from 27 to 47 units. In none of these did a dose of 40–50mg scoline cause apnœa lasting more than four minutes.

Hypotension.

Transient falls in blood pressure following full curarisation and controlled respiration are common in these cases as in other operations. Reversion to a level closely below the pre-operative figure occurs after an interval of about ten minutes. Additional doses of thiopentone, however, are likely to be attended by hypotension which may persist throughout operation. If anæsthesia is judged to be too light, intravenous pethidine 15–25mg is a more satisfactory choice and has only once caused a fall of pressure. Profuse hæmorrhage may occur at any time in these operations, so that massive rapid transfusion is required to correct hypotension. The MRC transfusion set is preferred to the new disposable one, as it permits faster pressure transfusion with less trouble. When using the plastic set, an artery forceps must be applied to the air-inlet every time the pressure in the bellows is raised and clotting in the filter chamber occurs more readily.

Hiccough

This irritating complication of the modern anæsthetic technique was observed frequently in the early cases of this series. It is now seen but rarely. It appears to be initiated by excessive hyperventilation in the absence of adequate analgesia, or more rarely, curarisation. Once started, it may most effectively be abolished by the administration of intravenous pethidine. If relaxation appears inadequate, a dose of 10mg tubo-curarine may be required in addition.

Excessive ventilation has been observed in these and other abdominal operations to mask the need for increments of relaxant. The true state of muscle tone will, however, be revealed during peritoneal closure, when removal of the retractors will be followed by rapid return of muscle power and extrusion of bowel. This, like hiccough, may be avoided by the regular intermission of controlled respiration in order to estimate the degree of intercostal paresis and the need for increment of relaxant. Analgesia is judged to be inadequate if limb movements or swallowing occur, or if the blood pressure and pulse rate are rising. Pethidine 10–30mg is preferable to and more logical in use than thiopentone. It is convenient to chart blood pressure and pulse rate at fifteen minute intervals in the absence of hæmorrhage, and to assess analgesia and relaxation as described above.

Analysis of cases

NUMBER OF PATIENTS		129
AGE	Eldest	73 years
	Youngest	2 years
	Average	38 years
SEX	Male	77
	Female	52
DIAGNOSIS		
	Cirrhosis hepatis	109
	Mild	34
	Advanced	75
	Extra-hepatic obstruction	20
	Cavernoma	13
	Other	7
OPERATIONS		
	Porta-caval anastomosis	74
	Lieno-renal anastomosis	33
	'Portal-systemic exchange' (arterialisation of liver)	4
	Splenectomy	6
	Proximal gastric transection	6
	Proximal gastric resection	12
	Exploratory laparotomy	10
	Ligation of varices	2
	Injection of varices	3
	Rienhoff's operation (hepatic arterial ligation)	4
	TOTAL	154

Anæsthetics

TECHNIQUE	
Thiopentone, nitrous oxide-oxygen ether, nitrous oxide-oxygen tubocurarine	28
Thiopentone, nitrous oxide-oxygen, tubocurarine	52
Thiopentone, scoline, nitrous oxide-oxygen, tubocurarine	70
Thiopentone, closed ether and oxygen	1
Thiopentone, scoline, nitrous oxide-oxygen, trichlorethylene (for injection of varices only)	3
TOTAL	154

DOSAGE		
Thiopentone		
Maximum		1000mg
Minimum		200mg
Average		620mg
Tubocurarine		
Maximum		90mg
Minimum		10mg
Average		53mg
Pethidine (excluding final dose)		
Maximum		100mg
Minimum (none given to 97 cases)		0mg
Average		50mg
Post-operative course (28 days)		
Uneventful Recovery		110
MORBIDITY		
Pulmonary collapse		8
Episodic stupor		5
Jaundice		4
Bronchitis		3
Pneumonia		2
Hæmatemesis		2
Paralytic ileus		1
Hæmothorax		1
Pulmonary embolism		1
	TOTAL	27=17%
MORTALITY		
Liver failure		7
Hæmorrhage		7
Pneumonia		1
Mesenteric thrombosis		1
	TOTAL	16=10%

(In the last three years the mortality has been reduced from 10 to 8 per cent)

DISCUSSION

Many of these patients were suffering from a grave illness, for whom the outlook, whether medical or surgical, was poor. No death occurred in patients with extra-hepatic obstruction or mild cirrhosis. The mortality occurred exclusively among those suffering from advanced cirrhosis and in such patients the capacity of the liver to survive a combined surgical and anæsthetic insult is at present impossible to assess.

Blakemore[6] favours cyclopropane as the anæsthetic of choice, but it was not employed in this series for a variety of reasons. The use of such an agent in operations where heavy blood loss is expected must prolong the operating time, since diathermy cannot be used. It produces poor abdominal relaxation in most cases, and to supplement it with relaxants makes the signs of depth impossible to interpret. In major abdominal and thoracic operations the abandonment of cyclopropane in favour of light narcosis with barbiturates and relaxants has produced a marked improvement in the condition of the

patient during and especially after, anæsthesia. Of the deaths, the pattern in respect of liver failure has been one of normal recovery from the anæsthetic before coma developed days later.

Hæmorrhage has usually been due to post-operative hæmatemesis when a shunt has been impossible or when it has become occluded. In two patients enormous blood loss occurred during operation and under-transfusion was almost certainly to blame for delayed death from liver failure. It is therefore imperative to replace blood as it is lost.

An attempt has been made in all cases to keep the anæsthetic level as light as reasonably possible, while avoiding excessive zeal in this direction. 'Sailing too close to the wind' has its dangers and it is debatable if narcosis so inconspicuous as to permit facial and limb movements, swallowing and hiccough is in the interest of the patient.

SUMMARY

An account has been given of the conditions causing portal hypertension and some operations employed for its relief. Special conditions affecting the anæsthetist are mentioned. An anæsthetic technique suitable for these operations is described in detail and the difficulties which may arise during anæsthesia outlined.

An analysis of 154 anæsthetics is included and the mortality and choice of technique discussed.

Acknowledgement

I wish to thank Mr Alan H. Hunt, surgeon, St Bartholomew's Hospital who performed all the operations for his tolerance in the theatre and for much help in the preparation of this paper.

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Halothane

Some difficulties and dangers in its administration

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Reports from a number of sources of the good operating conditions provided by halothane anæsthesia have led to its employment for a wide range of surgical procedures. Whether a closed or semi-closed apparatus is used, induction is quiet, and coughing or straining is uncommon even in bronchitic patients, provided that suitable premedication has been given.

The hypotension often induced by halothane contributes towards a relatively bloodless field, while its potency allows maintenance of adequate depth of anæsthesia without intravenous supplements of relaxants, pethidine or thiopentone ordinarily required during longer procedures. The high cost of halothane may be considerably mitigated by the saving on other drugs and this is particularly so when a closed circuit method is in use. If a closed circuit machine of the Marrett type delivering halothane-oxygen is employed and induction is commenced with not more than 250mg of thiopentone together with 0.3 to 0.6mg atropine sulphate in the fit adult, surgical anæsthesia is rapidly obtained and soon reaches a point where intubation is easy without recourse to relaxants[1]. Conditions will be found to be satisfactory for upper abdominal surgery, as there is full muscular relaxation with quiet respirations[2]. Carbon dioxide retention may accompany the deeper levels of halothane anæsthesia[3], as might be expected where poor tidal volume is present in a situation where control or assistance is on other grounds best avoided. Clinically however these patients do not appear in a less satisfactory condition post-operatively than those dealt with by the relaxant technique.

In spite of the good operating conditions and the apparently easy conduct of halothane anæsthesia, there are aspects of its clinical action which require further investigation before its ultimate position as a safe anæsthetic agent can be determined. The writer's experience is limited to some 500 administrations either alone or in collaboration

with the staff of four hospitals where the properties of halothane are being investigated clinically; so that clinical impressions rather than statistical proof are adduced in this account of its employment, which takes note of some of the difficulties which may be met with at various stages of anæsthesia.

INDUCTION

Apnœa and hypotension

In the fit adult the induction of anæsthesia with halothane following a small initial thiopentone injection will be found to produce a fall of blood pressure which is approximately proportional to the depth of anæsthesia. If the induction proceeds to a deep level, as for intubation in the halothane-oxygen circle method, there will be an uncomplicated fall of blood pressure to a low level. Sudden increases of concentration at this or any other time in anæsthesia may be accompanied by the development of undesirable arrhythmias. At a point just before apnœa supervenes, the pulse is scarcely palpable and the systolic reading may be as low as 60mm Hg. Respiration now ceases but immediate reduction of the halothane concentration to zero is followed after an interval of some seconds by the gradual return of respiration and a concommitant rise in blood pressure.

Apnœa due to deep anæsthesia is best treated by inflation of the lungs once or twice with pure oxygen – the procedure should then be repeated after a short interval[4]. Continuously assisted respiration, which will superimpose a lowering of carbon dioxide tension, further apnœa and an additional hypotension, is contra-indicated. Profound hypotension and apnœa, which may be worsened by over-assistance, is a potentially dangerous situation for the relatively inexperienced clinician. The experienced administrator will not make mistakes during induction, but may occasionally be 'caught out' during the operation when requested to deepen anæsthesia quickly. If there is a fair degree of tissue saturation – relaxation, apnœa and hypotension even as low as 40 to 50mm Hg may be obtained very rapidly.

Stress is laid on the possible dangers of halothane hypotension here in view of the role of thiopentone in causing anæsthetic accidents, many of which have been due to its hypotensive action[5 6]. Reversible hypotension is of the greatest value in the hands of the expert and in selected cases. The method of using the drug described above applies to the fit patient, but matters will be considerably complicated in the presence of such conditions as chronic hypertension. In such patients the fall of blood pressure at quite moderate depths of anæsthesia may be profound.

Case 1

A patient who was to be operated on for total laryngectomy exhibited a pre-operative systolic blood pressure of over 200mm Hg. Early in halothane-oxygen closed-circuit anæsthesia the systolic level fell to 60 to 70mm Hg where it remained for three hours. On substituting high flow nitrous oxide-oxygen (10 litres total flow per min) for securing bleeding points, it rose quickly to 125mm Hg.

In the case of hypotensives the fall in blood pressure tends to be much smaller relatively, but much care is needed in its control.

Case 2

A middle-aged alcoholic woman was found to have a pre-operative blood pressure of 95mm Hg systolic, which gave place under closed halothane-oxygen anæsthesia with some rapidity to a level of only 50mm Hg. During laparotomy and cholecystectomy, three injections of methyl-amphetamine of 10mg each and a slow drip of whole blood,

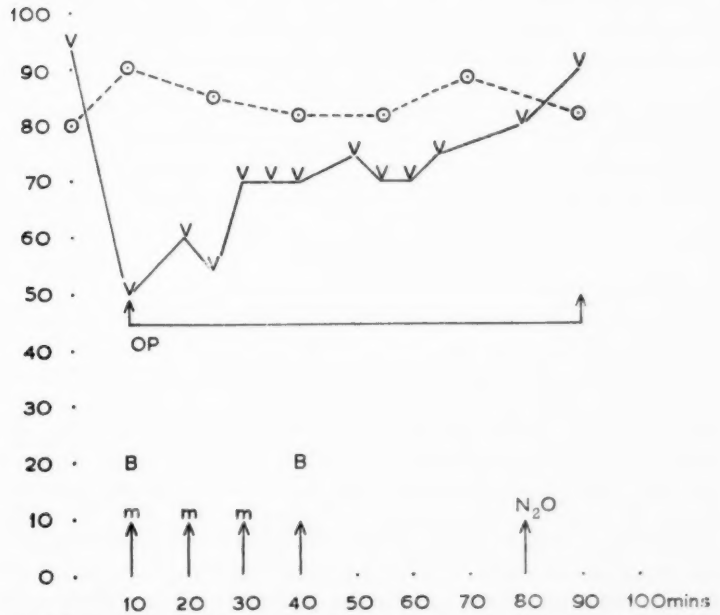

FIG. 1 Response to halothane-oxygen in the hypotensive patient. Female alcoholic aged 62. Pre-op BP=95/35. Hb=78 per cent. Marrett machine, fully closed, with basal oxygen flow, giving concentrations from 4 per cent in induction to 2.1 to 2.4 per cent in maintenance (Marrett, 1959). BB=500ml whole blood. mmm=10mg of methylamphetamine. OP=operation commenced. N_2O= halothane off, nitrous oxide-oxygen on (10 litres per minute semiclosed).

maintained the level at 70mm Hg. On ceasing the halothane, the level of pressure while on high rate flow of nitrous oxide–oxygen (10 litres total per min semi-closed) rose to the pre-operative level in a few minutes (FIG. 1).

Where falls of blood pressure are precipitous in frail patients it may be wise to abandon the drug. Severe hypotension is much more commonly seen using halothane-oxygen by the closed method. But an occasional patient will react in this way even when subjected to low percentages of halothane as an adjuvant to nitrous oxide-oxygen given at high flow rates. In contrast to the type of patient discussed above are those who, like the trimetaphan-resistant patient, fail to show even that moderate fall in pressure which would be expected with ordinary dosage.

Case 3

Such a response is well illustrated by the case of a young woman anæsthetised, intubated and maintained on halothane-oxygen (closed), for a partial thyroidectomy. The final stage of suture was carried out under nitrous oxide-oxygen (10 litres per min).

The overall picture in relation to the hypotensive effect of halothane is unpredictability. For this reason the careful anæsthetist will regard the hypotensive action of the drug with the same caution as he affords to that of thiopentone in the hypertensive and the ill and frail patient.

MAINTENANCE

Cardiac effects

Dobkin[7] considers that halothane 'undoubtedly causes a characteristic depression of the human heart', but McGregor[8] and his collaborators, while presenting impressive evidence of reduction of cardiac output in children under halothane anæsthesia, point out that diminished cardiac output in the presence of reduced oxygen uptake due to anæsthesia need not be interpreted as myocardial depression. Be that as it may, the most common source of anxiety during the maintenance period with halothane anæsthesia is the development of arrhythmias, the incidence of which would appear to vary with the site of operation, the depth of anæsthesia and the care with which they are sought by the administrator. These arrhythmias are attributed by Dobkin to a depression of conduction at the sino-auricular node 'so that ectopic centres discharge and nodal rhythm or ventricular extrasystoles are not uncommon. Ventricular arrhythmias are seen particularly if induction to a deep level of anæsthesia is rapid'.

Patients undergoing halothane anæsthesia may be divided into

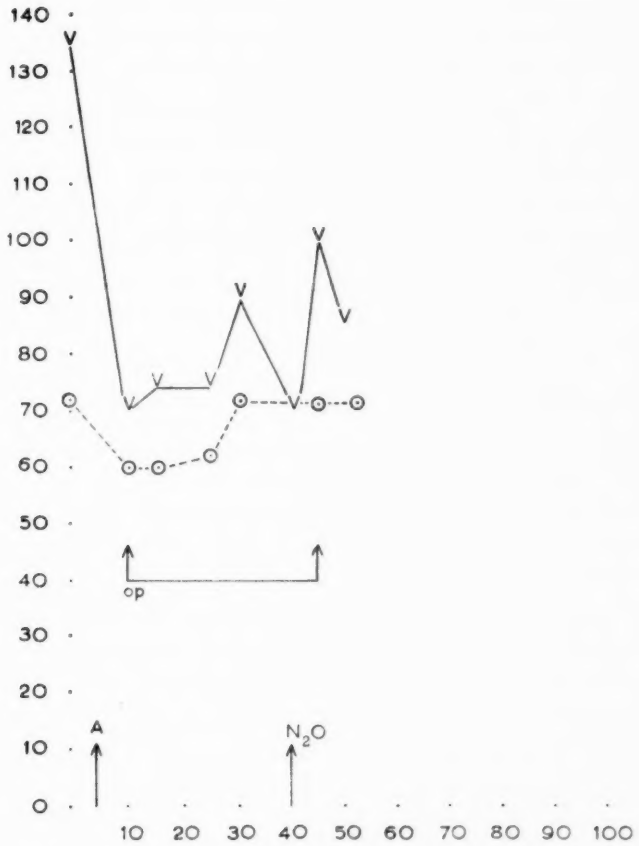

FIG 2A Typical hypotensive response to halothane–oxygen in a normotensive patient. Female aged 54. Initial bradycardia noted. No cardiac irregularities followed infiltration of cervical tissues with 1/200,000 adrenaline in saline. Pre-op BP= 135/85. Pulse 72. Partial thyroidectomy for nontoxic adenoma. A= adrenaline 375μg. op= operation commenced. N_2O= halothane off, nitrous oxide-oxygen on (10 litres per minute). VVV systolic BP. ○○○ Pulse rate.

four main groups—excluding those who are already the subject of cardiac disease: the larger group who show no irregularities; those where occasional extrasystoles are observed; patients with well marked runs of extrasystoles; those showing gross interference with normal rhythm such as fibrillation, or coupled beats. Many of the patients displaying arrythmias are otherwise fit and healthy.

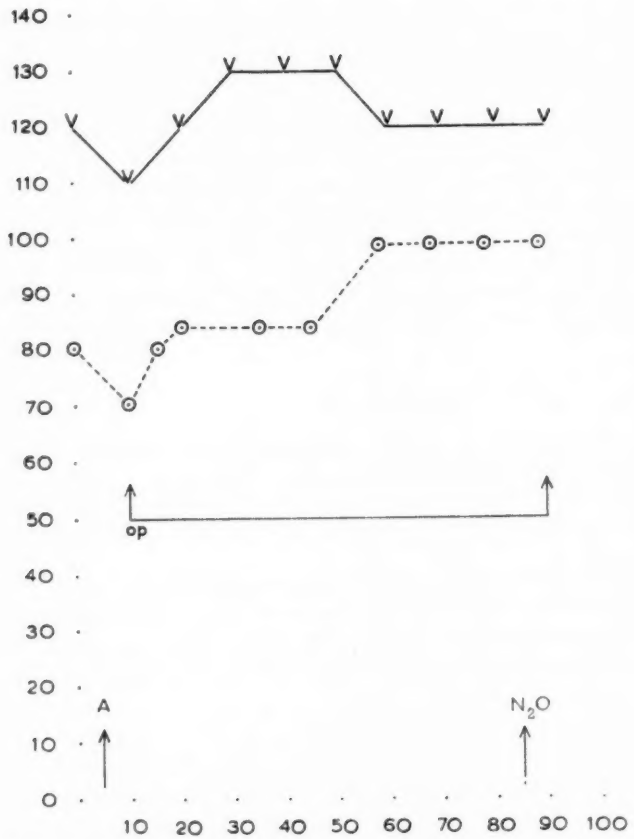

FIG 2B Illustrating a less common response to halothane-oxygen. Fall in blood pressure (10mm Hg) is insignificant. No cardiac irregularities followed cervical infiltration with 1/200,000 adrenaline in saline. Pre-op BP= 120/80. Pulse rate 80. Female aged 25. Partial thyroidectomy for thyrotoxicosis. A= adrenaline 500μg. N_2O= halothane off, nitrous oxide-oxygen on. op= operation commenced. Patients illustrated in FIG 2A and 2B were subjected to halothane concentrations varying from 4 per cent early in induction to 2.1 to 2.4 in maintenance (Marrett 1959) in a Marrett machine.

Case 4

Two male patients in the same operating list, both in early middle age, one undergoing removal of a small melanoma of the leg, the other a hernia repair, were anæsthetised by the closed halothane-oxygen method (Marrett machine), and exhibited extrasystoles of the group

two type above, progressing to group three during anæsthesia and disappearing within fifteen minutes of the withdrawal of the halothane.

Children anæsthetised with a Goldman[9] halothane apparatus with semiclosed nitrous oxide-oxygen for induction and a calibrated halothane bottle for maintenance have been observed to display a variety of arrhythmias during tonsil dissection. Short bursts or runs of extrasystoles are not uncommon in these cases, though the intermittent character of the arrhythmias is such that they will often only be detected by careful and prolonged observation during the operative period, and their clinical significance is somewhat doubtful. In the case of patients who are the subjects of disorders of rhythm there would seem to be good reason for avoiding halothane.

Case 5

A male patient of early middle age, with auricular fibrillation, well controlled on digoxin, was anæsthetised with halothane-oxygen by the Marrett method, following atropine and thiopentone. Early in anæsthesia he displayed a grossly irregular pulse including runs of very fast beats, which contrasted sharply with his condition when the halothane was abandoned and ether-oxygen substituted. Here the halothane appeared to have reversed the process of digitalisation.

It may be that in some patients the presence of endogenous circulating adrenaline may predispose to arrhythmias with halothane. Three patients, all nervous young women, were observed to display well-marked pulse irregularities under halothane anæsthesia. The most marked of these occurred in a young woman who had been exposed for a short period during induction to nitrous oxide-oxygen and minimal trichlorethylene. The question is raised by this case as to a possible undesirable synergistic effect of two halogen drugs acting on the pacemaker.

Case 6

A young woman of thirty years, an ex-SRN and doctor's secretary, was very nervous lest she be subjected to an inhalation induction. The method employed was atropine and thiopentone, followed by halothane–oxygen (closed) by Marrett machine. The operation was lymphangiogram in the Trendelenburg position. Well-marked pulse irregularities developed. Only an occasional extrasystole could be detected during the recovery period.

Case 7

A very nervous girl aged twenty-one developed a tachycardia in the anæsthetic room in spite of good premedication. The calibrated halothane bottle was not available for induction owing to an

unexpected delay in the preceding case. Anæsthesia was therefore induced with 500mg of thiopentone, 40mg of gallamine triethiodide, nitrous oxide-oxygen and the smallest possible trichlorethylene supplement. In the theatre she was breathing spontaneously and was then maintained with halothane nitrous oxide-oxygen during an operation for menisectomy. She soon developed well-marked runs of extrasystoles. After some minutes it was decided to abandon the halothane, and minimal ether was substituted. The irregularities disappeared but reappeared thereafter at intervals in a less accentuated form. She was kept near the theatre and an electrocardiogram done. This showed runs of extrasystoles of supraventricular origin with pulsus trigeminus (FIG. 3). This state of affairs persisted for an hour after

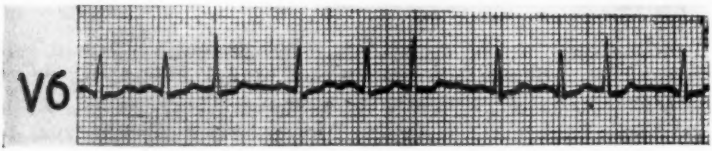

FIG. 3 Electrocardiographic tracing in a woman of 21 who developed persistant pulse irregularities during an operation for menisectomy (see text). Lead V6. Supraventricular ectopic beats, with pulsus trigeminus.

withdrawal of all anæsthesia. Seen next day in the ward, she reacted to the visit by developing a tachycardia of 120. The extrasystoles had disappeared. No clinical evidence of thyroid disease was found.

Case 8

A severe arrythmia occurred in an apparently fit woman of forty-nine years undergoing operation for the cure of recto-vaginal fistula. Induction was by 250mg of thiopentone, halothane-oxygen (closed), by Marrett machine. Hyoscine 0.4mg had been given, but not the atropine, 0.3 to 0.6mg of which is usually employed in addition to the thiopentone. She developed a bradycardia of 56 to 60, with no evidence of fall in blood pressure. Early in the operation the bradycardia gave place to what was clinically indistinguishable from fibrillation—an irregular irregularity with runs of fast beats up to 150 per minute and occasional long gaps when beats failed to reach the wrist. Atropine was given and halothane abandoned in favour of diethyl ether. Within five minutes the irregularity was replaced by an occasional extrasystole. She now had tachycardia due to atropine. Anæsthesia was maintained for a further seventy-five minutes by nitrous oxide-oxygen and minimal ether, with small supplements of gallamine triethiodide and pethidine. There was no reappearance of the arrythmia.

The above case illustrates the importance of giving atropine during induction, but the writer is by no means convinced that this precaution is necessarily a guarantee that arrhythmias will not occur. While there is evidence to relate an increased incidence of arrhythmias during maintenance of anæsthesia with halothane to such factors as surgical stimulation in light anæsthesia and the presence of injected or endogenous adrenaline, other causes may occasionally be of importance, such as a sudden increase in the concentration of the anæsthetic mixture.

Case 9

A small child was being operated on for tonsil dissection when the surgeon suddenly observed that the anæsthesia was becoming too light, and the child confirmed this observation by coughing slightly. The concentration of the halothane was suddenly increased and the electrocardiograph to which this patient had been connected for recordings during anæsthesia at once showed coupled ventricular ectopic beats. (FIG. 4).

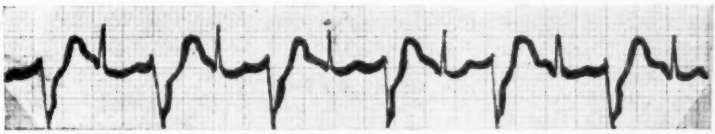

FIG. 4 Electrocardiographic tracing in a child of eleven years, during tonsil-dissection under nitrous oxide–oxygen halothane.

Coupled ventricular ectopic beats, with sinus rhythm. Apparently the direct result of a sudden increase in concentration of the drug. (Lead II.)

The case quoted above would seem to lend support to the view that halothane should not be administered in concentrations above an agreed safe figure. Hart[10] in a group of neurosurgical and other cases found that the incidence of arrhythmias was lowered by the use of an apparatus developing a maximal concentration of 1.5 per cent. Such an apparatus could not however produce conditions suitable for upper abdominal surgery, as such workers as Marrett and Ellis have done using closed halothane-oxygen. Marrett has conducted experiments to show that the concentrations in his machine at the mark $\frac{3}{4}$ of I do not in fact rise higher than 2.4 per cent, owing possibly to the continual destruction or removal of halothane by the tissues by a process not well understood[11]. Care should be taken during the use of the Boyle-Davis gag with halothane that sudden increases of concentration are not permitted; the old device of placing a swab over the

mouth should the anæsthesia lighten should never be employed with halothane, though it may be unobjectionable when ether is in use.

The relationship between raised blood adrenaline levels due to injected local analgesia solutions containing the drug, and halothane arrhythmias, is one which requires investigation, since the combination of local analgesia with an inhalation method is one which offers particular advantages in some fields, notably in the surgery of the head and neck. In their small series Brindle *et al*[12] reported a very high incidence of electrocardiographic changes amounting to nearly 50 per cent in the neurosurgical cases they investigated. But such changes were also noted frequently prior to adrenaline infiltration. Langdon[13] has investigated a group of seventy-six neurosurgical cases subsequent to adrenaline infiltration and reports some 20 per cent of electrocardiographic changes. It is of interest in view of these figures that Hurter[14] who has observed eighty neurosurgical cases subject to hypothermia and anæsthetised with nitrous oxide-oxygen halothane, but not subject to adrenaline infiltration, states that continuous ECG monitoring has revealed few changes in his series. Occasional ventricular extrasystoles in these patients have been noted, but they tend to be brought on by undue movement of the cooled, anæsthetised patient rather than by any other factor.

RESPIRATORY EFFECTS

During the maintenance period of anæsthesia halothane may occasionally present problems due to its respiratory effects. Apart from any central effect it may appear as a respiratory depressant in some patients owing to the speed with which it produces muscular relaxation. An occasional failure will be met with during halothane anæsthesia in patients who exhibit an undue sensitivity to central depressants used for premedication. Such patients may be impossible to maintain on halothane since they cannot be even adequately induced before apnœa occurs. Such an apnœa may have to be distinguished from that due to depth, which is usually accompanied by a well-marked hypotension. Such patients as have been described above could in theory be dealt with by control of respiration, but the advisability of this procedure is the subject of considerable disagreement. The writer's experience is that while control of respiration is unnecessary and probably dangerous when using halothane-oxygen in a closed circuit, it may be safe enough when the agent is being employed at low concentration as an adjuvant to a relaxant. In these circumstances halothane is not being employed in sufficient strength to secure relaxation unaided, but it is of value in producing a degree of controlled hypotension, in avoiding the use of supplementary drugs

such as pethidine or thiopentone, and in reducing the amount of relaxant needed. A method which gives good results in suitable cases is to employ a suxamethonium drip, which is easily controllable, and to maintain anæsthesia by means of a Boyle circle-absorber with a constant leak, with the halothane in the calibrated bottle on the machine above (not in) the circle, and a gas flow not exceeding 4 litres of combined nitrous oxide-oxygen. With this technique a suitable concentration of halothane can be obtained which will allow control of respiration without depressing the systolic blood pressure below 80 to 90mm Hg, and any further tendency to hypotension can be counteracted by shutting off the halothane temporarily.

The halothane-oxygen circle method, where control of respiration is not permitted, has its limitations. If abdominal relaxation is to be adequate there will be a degree of respiratory depression which, while permissible for the shorter procedures occupying 45 to 60 minutes, may be held to be undesirable for operations such as abdomino-perineal excision of the rectum, portal-venacaval shunts for the treatment of hæmorrhage in cirrhosis of the liver, or abdominal aortic grafts. These occupy a good deal of time and may be accompanied by extensive hæmorrhage at certain stages of the operations. On occasion deep anæsthesia with halothane–oxygen may be marred by tachypnœa which has supervened at a lighter level and which, though to some extent amenable to the influence of pethidine, remains persistent. The relaxant technique still tends to be the method of choice in extensive abdominal procedures where full relaxation is required, as not only is full oxygenation ensured by hand or machine, but undesirable carbon-dioxide 'build-up' is avoided, and the poor-risk patient spared the additional work of moving the thoracic cage during the operative procedure.

In children, using the semi-closed method, halothane tachypnœa does not seem to present much of a problem, and in them the quiet respiration and the absence of secretions may more than compensate for a slight increase in the respiratory rate. Goldman[9] reports some evidence that electrocardiographic changes in children may be related to actual surgical stimulation under what would otherwise appear to be an adequate level of halothane anæsthesia. These findings would tend to confirm those of Robson and Sheridan[15] who reported the relationship between a strong stimulus, such as stretching the rectal sphincter, and extrasystoles of ventricular origin.

RECOVERY

Recovery from halothane anæsthesia is often rapid and relatively smooth.

Case 10

An elderly woman subjected to an average concentration of the drug of 0.7 per cent for two hours during a rectal operation was able to answer questions before she left the theatre.

Patients of a muscular build often display a well-marked second stage, in which the musculature may be held in a state of extreme rigidity and tension, often accompanied by tremor. A few patients have been seen to exhibit an almost board-like rigidity, which may give rise to cyanosis and hypoxia due to the immobility of the chest wall, or to the involvement of the laryngeal muscles in the state of generalised spasm.

Case 11

A young man who had been satisfactorily anæsthetised with halothane-oxygen for a minor rectal operation exhibited extreme generalised muscular rigidity and laryngeal spasm after he had been restored from the lithotomy position. The condition was overcome with some difficulty by oxygen inflation; a few minutes later laryngeal spasm and general rigidity recurred, and he was again treated by oxygen inflation. Thereafter his condition gradually returned to normal.

There is a danger that the quiet and easy induction with halothane may lead to a false sense of security about the recovery period. A particular danger has been noted and reported by Young and Lodge[16] in patients anæsthetised by halothane-oxygen, who have been subject to nitrous oxide-oxygen during the final minutes of the operative procedure. Sudden collapse, often accompanied by cyanosis, may occur without warning at this juncture. The mechanism of this deterioration in the patient's condition is obscure, but it may be related to the collapse previously reported in patients on cyclopropane-oxygen following the cessation of the high percentage oxygen mixture. This condition of what may be termed 'halothane shock' may be particularly dangerous and lead to actual cardiac arrest if it supervenes when there is a halothane-induced hypotension.

SUMMARY AND CONCLUSIONS

In spite of the success achieved with halothane by skilled administrators, often with quite large series of patients, it is potentially the most dangerous inhalation agent to find wide employment since chloroform. The best results will be obtained by a very careful anticipation of untoward events at all stages of anæsthesia. During induction, atropine (0.3mg) and a reduced dosage of thiopentone (not

more than 250mg in the fit adult if closed halothane-oxygen is to be employed) will tend to minimise bradycardia and hypotension. The correct handling of simultaneous apnœa and hypotension should be envisaged. Undesirably low or prolonged hypotension may usually be corrected by methoxamine or methylamphetamine, provided that reversed Trendelenburg position is dealt with by suitable change of posture and blood loss by transfusion.

Observation of the blood pressure level and cardiac rate and rhythm acquires a new importance with this agent, as diminution of cardiac output and depression of the conducting fibres are not infrequent. Full relaxation can be obtained with halothane-oxygen in a circle inhaler; its use in prolonged cases is inadvisable owing to reduced tidal volume and tachypnoea giving rise to carbon dioxide retention. The danger of sudden collapse following the withdrawal of halothane-oxygen and the substitution of nitrous oxide-oxygen should be envisaged and the oxygen percentage in such a mixture should be as high as 60 per cent at first and gradually lowered. Special care should be taken with the nervous patient. Arrhythmias may be met with more frequently in such patients and in the child subjected to surgical stimulation under light anæsthesia; the possible role af endogenous circulating adrenaline should be envisaged. Some investigators have employed concentrations of adrenaline for infiltration of not more than 1 in 200,000 without ill effect in the presence of halothane and this is the writer's experience. Others however condemn the practice. Cardiac arrhythmias tend to be induced by sudden increases of concentration of halothane, and such increases should be avoided. Existing fibrillation may be worsened by halothane. Gross arrhythmias should be counteracted by withdrawal of halothane in favour of some agent of a non-halogen type. If controlled respiration is used with halothane, a low percentage (0.4 to 0.7) and the use of a suxamethonium drip for relaxation would seem to be the safest method, while careful watch is kept on the level of the systolic blood pressure. Care should be taken when using the Boyle-Davis gag, that a high concentration of halothane is not built up by covering the mouth in an attempt to deepen anæsthesia rapidly. In the recovery period a few patients will show an unduly intense muscular spasm and rigidity, leading at times to laryngeal spasm, and a careful watch should be kept for this condition. The condition of 'halothane shock' which may follow the withdrawal of high oxygen percentage mixtures may be delayed well into the recovery period and patients should be watched with more than usual care therefore.

Johnstone[17] states that the occurrence of cardiac arrhythmias 'reflect more on the management of the patient than on the drugs used'. While not in full agreement with this statement the writer has come to

regard halothane as a substance which is liable to give rise to serious difficulties with even small errors of judgement and it is perhaps more than a mere verbal quibble to ask whether such an agent—while undoubtedly of great value—may be described at this stage as safe.

Acknowledgements

I am much indebted to Drs Frankis Evans, George Ellis and Victor Goldman for access to clinical material at St. Mark's Hospital, St Bartholomew's Hospital, and the Battersea General and Queen Mary's Hospitals, and for valuable advice. My thanks are due to Dr J. Wedgewood, First Assistant in the Department of Cardiology, St. Bartholomew's Hospital, for electrocardiographic reports.

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Halothane and hypotension

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The first pharmacological studies of halothane by Raventos[1] established that this anæsthetic agent has a powerful hypotensive action. The cause was ascribed to selective ganglion paralysis affecting the mesenteric ganglia because it was found that the superior cervical ganglia were not readily paralysed. Depression of cardiac output by weakening the cardiac muscle was not considered an important cause of the hypotension. Johnstone[2] explained the bradycardia and hypotension as due to depression of sympathetic activity with sparing of the cardiac parasympathetic mechanism and described other signs of autonomic depression such as persistant vasodilatation, the absence of sweating, the suppression of salivary, bronchial and gastric secretions and the absence of vomiting and retching in the recovery period. Brennan *et al*[3] considered hypotension was due to inhibition of sympathetic activity with activation of the cardiovascular parasympathetic innervation. The hypotension was further investigated by Burn *et al*[4] who performed experiments to determine the action of halothane on cardiac output and ganglion activity. Cardiac output in the dog heart-lung preparation was depressed by halothane, particularly with a vapour concentration of 2 per cent. It was demonstrated that halothane lowers the blood pressure in the cat under chloralose to a greater extent than can be readily accounted for by ganglion block and that it lowers blood pressure in the animal in which the splanchnic vascular bed has been removed by evisceration. It was also shown to have a strong potentiating action on the ganglion block of hexamethonium and d-tubocurarine. From their experiments it was concluded that the ganglion blocking action of halothane could not account for its hypotensive effect, and that some other explanation should be sought for.

INVESTIGATION

These various reports and theories of the hypotensive action of halothane led to an investigation of its ability to produce controlled

hypotension for head and neck surgery in a maxillo-facial and plastic surgical unit. Firstly, it was used as the sole hypotensive agent in an attempt to reproduce conditions usually obtained by ganglion blocking drugs and posture. The posture of the patient was maintained as before, but it proved difficult, even impossible in many patients, to achieve a hypotension comparable with ganglion paralysants. Some reduction of pressure was often observed and at times a reasonably satisfactory operation field obtained, but it was seldom comparable with that achieved by ganglion blockade and a similar posture. In a few of the early administrations the concentration of halothane was further increased in an attempt to achieve a comparable hypotension. This could be obtained in some patients but not in all. These patients were slow to recover consciousness and the return of vascular tone was likewise delayed and unsatisfactory.

It was then used in association with ganglion blocking drugs when it was observed at once to potentiate their hypotensive effects. At the same time it was observed that the hypotension changed some of its characteristics and a fuller investigation was undertaken to determine more exactly the action of halothane. The following experiment was performed:

A male aged fifteen years was anæsthetised with thiopentone 450mg, suxamethonium chloride 80mg, C10 4mg and maintained on nitrous-oxide and oxygen (5:2) with a small flow (20ml.) of cyclopropane added on a closed circuit machine with constant leak at the valve. At the end of a one hour's operation the blood pressure with the patient horizontal was 160mm Hg systolic, with natural respiration. A steep foot down tilt of 25° produced no change of blood pressure. (160mm Hg). Suxamethonium 50mg was now administered and artificial ventilation continued with the same gas mixture as before. The blood pressure now was 145mm Hg in the same 25° foot down tilt and 160mm Hg when returned to horizontal again. Halothane 2.5 per cent was now admitted to the closed circuit (with the patient horizontal) and after respiring for three minutes the blood pressure in the horizontal position was 140mm Hg. A return to the same steep foot down tilt of 25° produced a small change only in blood pressure to 130–135mm Hg. When horizontal again the blood pressure remained at 130-135mm Hg and the patient was now encouraged to breathe naturally. When natural respiration had been resumed the blood pressure with the same concentration of halothane was 140-145mm Hg with the patient horizontal and with the same foot down tilt fell slightly again to 135-140mm Hg.

These figures demonstrate that halothane does not induce a posture sensitivity in the human subject and, therefore, presumably

does not block autonomic ganglia, a fact which has been further conclusively proved by repeating the above experiment on a patient immediately before operation and then proceeding to a fully controlled hypotension by the addition of a short acting ganglion paralysant. When this was done the blood pressure fell very considerably, especially in the foot down position, although it had failed to do so to any degree with halothane alone.

These experiments afford support to the findings of Burn *et al*[4] that the ganglion blocking action itself does not account for the hypotension induced by halothane. It is interesting to speculate, therefore, whether there is any central or peripheral action on the vascular system, or whether myocardial depression is sufficient to account for the observed hypotension. In concentrations of 2 per cent or more halothane causes considerable depression of the myocardium of the dog. It seems reasonable to assume that in the human a similar process occurs with high concentrations, whilst smaller concentrations cause hypotension by potentiating ganglion block. There still remains, however, a possible peripheral anti-adrenaline dilator action of halothane on the blood vessels as the cause of the observed hypotension. Burn[5] has recently shown by cross circulation experiments in dogs that halothane has a powerful relaxant action on smooth muscle. Halothane in 1.5 per cent concentration caused a fall in arterial resistance in the vessels of the dog's leg from 170–132mm Hg in one trial and from 153–119mm Hg in a second. This fall was accompanied by an increased venous outflow and when halothane was stopped the arterial resistance once more rose. These results could not be achieved by a 6 per cent concentration of ether and 8 per cent caused a fall of only a few millimetres. Thus Burn has provided experimental proof of the anti-adrenaline dilator action of halothane which works by a relaxation of smooth muscle in the wall of the blood vessels thus causing hypotension. For this reason intravenous vasoconstrictors do not have the same marked pressor action in the presence of halothane. Likewise the hypotension of halothane does not respond to respiratory and postural controls in the same way and this explains the differences observed in the early administrations.

TECHNIQUES

Halothane is a powerful hypotensive agent and although it may sometimes be used alone for the production of controlled hypotension this is not recommended. It has been used as an accessory hypotensive agent in association with ganglion paralysants in 1000 operations and now occupies a position of considerable importance in this technique. It has been used in the following ways:

As an anæsthetic prior to the use of ganglion blocking drugs.

Here the halothane is administered after induction and intubation under thiopentone and relaxant. A known percentage (1–3 per cent) is fed into a semi-closed circle absorber receiving a total gas flow of about 3 litres per minute, with constant leak at the expiratory valve. The full effects of halothane alone are observed with the patient prepared and positioned in a reverse Trendelenburg tilt immediately prior to the start of surgery. The hypotension achieved at this point is very seldom satisfactory and a fall lower than 80–100mm Hg is exceptional. Moreover the initial hypotension is seldom maintained after the surgery has started and the operation thereafter is usually performed at normal or only slightly subnormal blood pressures. But an adequate hypotension can now be achieved very satisfactorily by the administration of a short acting intravenous ganglion paralysant such as phenactropinium or trimetaphan. At this stage the body is remarkably sensitive to such drugs and small doses of each (5–10mg) are sufficient to produce a considerable fall of blood pressure. Because of this sensitivity a dangerously low blood pressure can be induced easily, and this cannot be reversed quickly. Bradycardia is often observed during induction with halothane but no additional atropine is administered because normal rate is always restored by the injection of the ganglion paralysant. Halothane has been used in this way in 200 patients and is the method of choice where the duration of hypotension is short and a rapid return of autonomic control is desired. It is also a great advantage in the older patient in whom the longer acting ganglion paralysants sometimes cause a prolonged and excessive hypotension.

As a subsidiary agent after ganglion paralysis has been established

Here a similar anæsthetic technique is followed but the hypotension is induced by ganglion block (with a drug such as pentolinium) followed by a reversed Trendelenburg tilt until the required hypotension is achieved. Halothane is administered when the blood pressure starts to rise and cannot be easily controlled otherwise. At this point it produces a most dramatic potentiation of the ganglion paralysis with a powerful and sustained hypotensive action, which can be repeated time after time if desired. This effect is often observed with very low percentages (sometimes less than 0.5 per cent) of the vapour, which must, therefore, be administered cautiously to avoid excessive and prolonged hypotension. This potentiation of ganglion paralysis is particularly valuable in those patients in whom the initial effects of ganglion blocking drugs and posture alone do not easily produce an adequate hypotension. Here again a low vapour concentration quickly causes a smooth and satisfactory fall of blood pressure which can

thereafter be maintained at the desired level with ease. In all patients with paralysed ganglia, high concentrations of halothane quickly cause a marked and prolonged hypotension which can only be reversed by excretion of the drug—a slow process, relatively speaking, and, therefore, dangerous and difficult to manage.

Halothane has been used in this manner in 800 patients and marked bradycardia has never been observed. No extra atropine has been given at any time. The standard practice of papaverine and scopolamine or morphine and atropine as premedication has been followed. Bradycardia does not occur when ganglion paralysants have been administered first. The extremely small concentrations of halothane vapour required in this technique makes it the most satisfactory and the most economical method of use, which is an important aspect in the case of such a particularly expensive agent.

As a result of this experience the following advantages and disadvantages of halothane in controlled hypotension can be stated.

ADVANTAGES

This is an agent with a different site of action—it is not a ganglion paralysant, even though it potentiates the action of such drugs. It continues to have an effective action when other hypotensive drugs have ceased to act or are only partially effective.

The hypotensive effect of all ganglion blocking drugs is short lived, in all probability because of the secretion of adrenaline, which results in a quick return of blood pressure to almost normal levels even though the ganglia remain fully paralysed. Halothane at this time has a marked hypotensive effect because of its direct action on the smooth muscle of the arterioles thus antagonising the action of adrenaline as well as potentiating ganglion paralysis. Halothane continues to exert its action for as long as its percentage is maintained in the inspired gases, the blood pressure rising when it is excreted and falling again when an increase is made. Thus halothane steps in with an effective measure of control, easily obtained and maintained, at a time when the human body is rapidly becoming resistant to all other hypotensive controls.

Without halothane it is necessary to induce hypotension with a large dose of ganglion blocking drug and to make sure that the minimum pressure is quickly obtained and most carefully maintained in order to exercise full control. It is possible now to use much smaller initial doses of ganglion paralysants and to induce the hypotension more slowly in the certain knowledge that with care an adequate fall of pressure can be obtained and maintained in every patient. It has been demonstrated that the rate of fall of blood pressure rather than

the minimum level is responsible for undesirable changes in the electro-encephalogram[6] and here is evidence in support of the claim that halothane enables controlled hypotension to be employed with greater safety.

DISADVANTAGES

When hypotension is induced with ganglion blocking drugs and posture it can be quickly reversed by changes of posture and respiration. This does not apply when high concentrations of halothane have been used. Posture then no longer causes such variations in pressure, and similarly respiratory control does not affect blood pressure to the same extent. There is now a new 'feel' to the hypotension. Any undue hypotensive action cannot be as quickly reversed because it must await excretion of the halothane by the lungs and three to five minutes are necessary for this depending on the concentration in the body. Consequently accurate control of blood pressure is more difficult until experience has been obtained and marked hypotension can be encountered easily.

There is very little vasoconstrictor action from an intravenous injection of any vasoconstrictor drug in the presence of high concentrations of halothane.

METHODS OF ADMINISTRATION

Halothane, as an adjunct to controlled hypotension, has always been administered in a closed circuit apparatus with carbon dioxide absorption. The constant leak technique with a gas flow of approximately 3 litres per minute has been employed and the halothane vaporised outside the circuit and fed in from some suitable apparatus. The early administrations were performed with a carefully calibrated vaporiser employing temperature control, but it soon became evident that accurate control of this nature was unnecessary. It is important to have a steady control of vapour concentration from minimal up to about 4 per cent (maximum) which can be obtained from the simple adaptation of the trilene bottle on the Boyle Machine (BOC pattern) or from the standard trilene bottle provided the plunger is kept fully elevated. A water jacket prevents the fall of temperature of the halothane liquid and ensures a more steady vapour concentration, but is not in any way essential. A thermometer in the halothane shows any marked change of temperature which is valuable information, but the temperature of the liquid which usually starts at theatre temperature always falls thereafter as the cold gases pass through the bottle. With relatively simple and cheap apparatus (on one machine only a modified chloroform bottle) a flow of halothane vapour is fed into the

circuit and the amount administered is regulated in accordance with its anæsthetic and hypotensive effects. More recently a change has been made to the Fluotec vaporiser. This has achieved a more accurate control of the vapour concentration which cannot be increased above 3 per cent but the response of the patient is the most important observation and the amount of halothane must always be adjusted to this and not to any fixed percentage. The vapour strength is increased until a satisfatory blood pressure control is achieved and it is then maintained at a steady concentration thereafter. It is possible when the hypotensive effect has been obtained to close the circuit completely by restricting the gas flow and maintaining the existing concentration in the circuit by basal flows only. This is a successful technique with the advantage of economy. On the other hand when halothane is used prior to ganglion paralysis a steady vapour concentration of 1–3 per cent is required, thus a more carefully calibrated vaporiser is an advantage. In both techniques care must be taken to avoid high concentrations of halothane which are particularly dangerous and any vaporiser should have an upper limit accurately set and calibrated for changes in temperature together with a smooth control of the range of vapour concentrations.

CONCLUSIONS

This technique has been employed primarily in controlled hypotension, but several conclusions can be made. Whenever halothane is administered a careful watch must always be maintained on pulse and blood pressure. By the use of an oscillometer (or some similar device) ill effects such as marked hypotension or bradycardia can be observed immediately and the vapour concentration reduced or adjusted accordingly. This careful approach to a valuable agent has many advantages over the arbitary percentage control of carefully calibrated machines. Ill effects can be encountered with minimal concentrations of halothane in some patients, especially those with paralysed ganglia and thus the safety and value of this new drug can be increased considerably by maintaining a watch over those aspects of the cardio-vascular system (pulse and blood pressure) most likely to be disturbed. The same technique is used when controlled hypotension is not employed, the concentration of halothane being adjusted to avoid ill effects on the pulse or blood pressure, thus increasing the safety of this useful and potent agent.

SUMMARY

Hypotension with halothane has been investigated in a maxillo-facial unit.

Its ganglion blocking action does not account for its hypotensive action, which it appears is achieved mainly by peripheral dilatation of the blood vessels.

Used in conjunction with ganglion blocking drugs it is a potent and valuable hypotensive agent.

High concentrations of halothane are markedly hypotensive probably because there is in addition a direct depression of the myocardium.

Wherever halothane is used constant attention must be given to both pulse and blood pressure.

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Post-operative postural hypotension

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'Significant post-operative hypotension is relatively common'[1]. In a series of 5,463 patients, 180 (one in thirty-one) were hypotensive during the recovery period following surgery[2]. In 176 unselected cases of major vaginal surgery, post-operative hypotension of significant degree occurred in 101 patients (57 per cent)[3]. A fall in blood pressure on return to the horizontal was found in fourteen out of thirty-one patients undergoing vaginal procedures[4].

Hypotension is more common following return to the horizontal position from the lithotomy position. In this paper, a series of seventy-five patients undergoing perurethal prostectomy is reviewed. These patients were operated on in the lithotomy position and thirty-seven (49 per cent) showed significant hypotension on return to the horizontal position. For the purposes of this article significant hypotension is a fall of blood pressure to a level 100mm/Hg systolic or to a level 50mm/Hg below the pre-operative systolic pressure.

ANALYSIS OF THE CASES

Age

The age distribution of the thirty-seven patients showing post-operative hypotension is given in TABLE 1.

Table 1

AGE	41–50	51–60	61–70	71–80	81 and over
NO.	1	8	11	16	1

The age distribution of the thirty-eight patients who did not show significant hypotension is given in TABLE 2

Table 2

AGE	41–50	51–60	61–70	71–80	81 and over
NO.	2	8	13	13	2

Duration of operation

The duration of operation in the hypotensive group is given in TABLE 3

Table 3

DURATION IN MINUTES	0–15	16–30	31–45	46 and over
NO.	1	8	16	12

The duration of operation in the non-hypotensive group is given in TABLE 4.

Table 4

DURATION IN MINUTES	0–15	16–30	31–45	46 and over
NO.	4	15	13	6

Premedication

Morphia was given in the premedication of twenty-three cases who developed hypotension and of twenty-three cases who did not develop hypotension. The remainder received atropine only.

Muscle relaxants

Gallamine triethiodide was the relaxant used. In the group who developed hypotension twenty received the relaxant whereas in the group who did not develop hypotension eleven received the relaxant.

Depth of anæsthesia

In twenty of the patients who developed hypotension the depth of anæsthesia was considered to be insufficient as indicated by movement, coughing, straining, or breath-holding; of these sixteen were given gallamine triethiodide to produce satisfactory operating conditions. In twelve of the patients who did not develop hypotension the depth of anæsthesia was insufficient using the above criteria and five of these were given gallamine triethiodide.

Blood during operation

Eighteen patients who developed hypotension received a blood transfusion during operation. In all of these cases the operation took forty minutes or more. Five of the patients who did not develop hypotension received a blood transfusion during operation. In one case the operation took thirty minutes and in the other four the operation took fifty minutes or more.

Anæsthetic agents

Sixty-five patients received thiopentone, nitrous oxide-oxygen, and trichlorethylene with or without gallamine triethiodide; five of these

also received pethidine. One patient had spinal analgesia. Nine had thiopentone, nitrous oxide-oxygen and pethidine with or without gallamine triethiode and with or without carbon dioxide absorption by soda lime. Patients who developed hypotension and those who did not were equally divided as regards the above anæsthetics and techniques.

Pre-operative blood pressure and blood pressure during operation

The pre-operative systolic blood pressure in the patients who developed hypotension is shown in TABLE 5.

Table 5

BLOOD PRESSURE	130 or below	131–145	146–160	161–175	176 and over
NO.	6	4	11	6	9

In one case the pre-operative blood pressure was not noted.

The pre-operative blood pressure in the patients who did not develop hypotension is shown in TABLE 6.

Table 6

BLOOD PRESSURE	130 or below	131–145	146–160	161–175	176 and over
NO.	9	6	12	5	6

During the actual operations about half of the patients in both groups showed a rise in blood pressure and about half showed a fall.

CAUSATION OF HYPOTENSION

Although hypotension, when it developed in this series, occurred almost immediately following the return to the horizontal postion, there is little doubt that the change in posture is not itself the main factor. One would expect that a patient with an intact vasomotor system and a normal blood volume would compensate adequately for a change in posture. Fairley, after a long discussion of the factors involved, did not consider change in posture of major importance[3].

From the analysis of the patients in this series, it would appear that the duration of operation, the depth of anæsthesia, the use of muscle relaxants and blood loss are the significant factors in post-operative hypotension. Age does not appear to be of significance but pre-operative hypertension may be of importance. Changes in blood pressure during operation do not appear to be of significance.

The use of morphine in premedication does not appear to affect the blood pressure in this series although the ability of morphine to produce postural hypotension has been noted[5].

The anæsthetic agents and techniques do not appear to be significant in this series. Hypotension has been noted after all types of anæsthesia[4].

Although the anæsthetic agents and techniques do not appear to be significant, there is little doubt that the patients were liable to carbon dioxide retention because of the restriction of respiration caused by the lithotomy position and the partial or complete depression of respiration caused by the use of muscle relaxants. Carbon dioxide retention is a well-known cause of post-operative hypotension[6]. The use of a carbon dioxide absorption technique or the 'wash-out' of carbon dioxide by hyperventilation did not seem to affect the incidence of hypotension in this series. It is notoriously difficult to estimate carbon dioxide content of the blood without laboratory analysis and it is possible that carbon dioxide retention may have occurred.

Fairley also discussed the occurrence of carbon dioxide imbalance, but did not consider it a significant factor in his series[3]. The duration of operation is obviously linked with blood loss, as the longer the operation takes the greater will be the blood loss. Nowadays there is a tendency to minimise the time factor because of the alleged non-toxic nature of anæsthetics and of the restorative measures used in long operations. However, reflexes from the operation site may produce adverse effects[7] and it has been pointed out that true blood volume loss may be greater than that estimated at operation[8].

The depth of anæsthesia and the use of muscle relaxants are also correlated. Both light and deep anæsthesia can cause hypotension the former by insufficient protection from noxious stimuli and the latter by depression of the cardiovascular system. Muscle relaxants by producing profound relaxation may cause pooling of the blood in addition to upsetting the carbon dioxide balance. Both factors can produce a fall in blood pressure.

A pelvi-cardiac reflex in vaginal surgery causing hypotension has been postulated[3]. In this series the filling and emptying of the bladder may produce a similar reflex especially during light anæsthesia. The use of distilled water in perurethral prostatectomy may cause hæmolysis with resultant hypotension[2]. The surgeons in this series use isotonic dextrose solution so that hæmolysis is unlikely. A solution containing penicillin, sulphacetamide, amino-acridine and thrombin is used at the end of the operation and it is possible that this may produce hypotension by a sensitivity reaction. However, the solution is not used in all cases and the occurrence of hypotension was not related to its use.

Although various factors can produce hypotension one can detect two main ways by which these factors act. These are by changes in

the vasomotor system or by fall in blood volume. One factor may act in both ways. For example a long operation may produce a fall in blood volume and vasomotor changes.

Lowering the patient's legs when either the blood volume or the vasomotor system is affected or when both are affected will now precipitate frank hypotension because the body is unable to compensate normally. The effect and the treatment of hypotension depends on whether blood loss or change in the vasomotor system predominates.

EFFECTS OF HYPOTENSION

The clinical picture of the patient suffering from hypotension in this series varied from profound shock with impalpable pulse and cessation of respiration or Cheyne-Stokes respiration to a condition where if the blood pressure had not been taken the patient would not have been considered to have hypotension. These patients were pink, dry and warm, had an easily palpable pulse of normal rate and were conscious and co-operative. These patients fall into the group where the vasomotor system is primarily affected whereas the patients who show signs of shock are those who have suffered primarily a fall in blood volume.

Fortunately, none of these patients appeared to have suffered permanent ill-effects although they have caused a good deal of anxiety. Cases have been described where permanent ill-effects have followed hypotension[9]. There is little doubt that the duration of the hypotension is of paramount importance. This makes it essential to detect hypotension early and the fortunate results in this series are probably due to early detection and to prompt and adequate treatment.

The patient who shows obvious signs of shock is not usually missed, but the case who does not, may be missed and suffer consequently. This can be illustrated by two cases who, on return to the ward, were propped up to encourage drinking. Both patients lost consciousness and became pale and shocked. Laying the patients flat quickly improved the situation and demonstrated clearly the dangers which beset patients whose vasomotors systems have been upset but who show no classical signs of shock.

No difference in urinary output or convalescence has been detected between the two groups.

All patients who have a prostatectomy are seen several months later and none appears to have suffered as a result of hypotension. There were five deaths in this series, three in the hypotensive group and two in the non-hypotensive group. None of these deaths could be attributed directly to hypotension.

PREVENTION AND TREATMENT OF HYPOTENSION

Unless routine recording of the blood pressure is performed there is little doubt that many patients suffering from post-operative hypotension are missed[1].

In view of the factors outlined above, which have been shown to predispose to hypotension, light premedication should be used and adequate depth of anæsthesia maintained without the use of relaxants unless needed. Especial care should be taken with those who have hypotension and blood replacement must be adequate.

Lowering and raising the legs repeatedly has been advocated in attempt to stimulate the vasomotor system[4], as has been the wrapping the legs and thighs with elastic bandages prior to lowering the legs[10].

If hypotension does occur, the reaction of the patient dictates to some extent the treatment to be followed. Where profound shock develops, rapid transfusion of blood, lowering of the head, and the giving of oxygen must be instituted without delay. The risk of overloading the circulation is slight provided the rate of the drip is slowed down when the blood pressure has reached 100mm/Hg systolic[11]. In less shocked patients blood is also indicated if the operation is prolonged. Hypertensive drugs are also of value.

Patients who do not show signs of shock one is tempted to treat conservatively. In cases with a slow pulse atropine may be given to abolish vagal overaction[12]. An intravenous drip should be set up in case the situation should deteriorate and the patient kept flat. If possible the patient should be kept in a recovery room. Everyone should be aware of the potential dangers and especially the risk involved in propping up these patients.

SUMMARY

Thirty-seven out of seventy-five cases undergoing perurethral prostatectomy showed a significant hypotension on return to the horizontal position at the end of the operation.

An analysis of the cases showed that the duration of the operation, the depth of anæsthesia, the use of muscle relaxants and blood loss are significant factors predisposing to hypotension.

Other factors are reviewed and the view is put forward that essentially blood loss or upset of vasomotor control or both are the underlying ways whereby hypotension occurs.

The effects of hypotension depend on whether blood loss or upset in vasomotor control predominates.

No permanent ill-effects have occurred in this series and this fortunate result is attributed to early recognition of hypotension and effective and rapid treatment.

Treatment depends on whether blood loss or upset in vasomotor control is predominant. Careful observation of the patient, replacement of blood loss and keeping the patient flat are vital points in the treatment of post-operative hypotension.

Acknowledgements

I wish to express my thanks to Dr I. M. Campbell Dewar, Senior Anæsthetist and to Mr T. L. Chapman and Mr J. W. Sutherland, Urological Surgeons, Victoria Infirmary, Glasgow for their encouragement in the preparation of this paper.

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Total bronchospasm and lung massage

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While partial bronchospasm is relatively common in anæsthetic practice, total bronchospasm is extremely rare. It is well known that bronchospasm can end in death[1] and that not only bronchial asthma, but any chronic disease of the lungs predisposes the patient to its development. Thiobarbiturates have been known to produce it, as well as tubocurarine. Neostigmine is extensively used experimentally to produce bronchospasm in animals and can be speedily removed by injecting atropine[4]. An anaphylactic-induced bronchospasm is effectively removed by isoproterenol[5] administered in ærosol. Much work has been done on this subject in connection with bronchial asthma. There is however this essential difference between the bronchospasm that is common in bronchial asthma and the total bronchospasm in anæsthesia. The former is seldom total, or else most of our asthmatic patients would be dead after their first attack. Death during an attack of bronchial asthma is known to occur, but such cases are extremely rare.

We have seen three cases of total bronchospasm during the past three years in a series of thoracic patients numbering over 700 individuals – an unusually high proportion.

Two of these developed in young men suffering from mitral stenosis, one of whom had chronic bronchial asthma as well. One case was seen in a man operated upon for pulmonary carcinoma under local analgesia. Of the three cases seen two had died on the table, the third case had his lung, as well as the heart, massaged and survived.

Our first case had advanced degree of mitral stenosis and chronic bronchial asthma. As he was very apprehensive we had decided to premedicate him with the usual lytic cocktail M 1, which provoked a violent asthmatic attack during the very moment of injection. It is at this juncture that we had been able to note *total silence* in the chest in spite of the fact that the diaphragm, the intercostal muscles and the accessory muscles of the neck were all actively engaged in an effort to carry on the respiration. This

condition ceased in a few minutes and we decided to induce sleep with thiopentone the next morning without telling the patient. The next day, during induction the patient developed a violent bronchospasm. Ventilation after intubation was impossible. As the heart stopped the chest wall was opened to find a grossly distended lung which would not collapse and an arrested heart. Heart massage was resorted to without producing any effect.

In this case our first diagnosis was poor relaxation due to some fault of the relaxant used, then obstruction of the endotracheal tube and finally bronchospasm. Injections of atropine, ephedrine and adrenaline were ineffective. Autopsy revealed the presence of several layers of hypertrophied muscle fibres around the bronchioles typical of chronic bronchial asthma.

Keating stresses that any chronic disease of the lungs may be a contributing factor in the development of bronchospasm. The truth of this assertion was borne out in our second case.

A man of 50 was operated under local analgesia (Prof. E. S. Lushnikov) for carcinoma of the lung. The tumour was found sitting astride the left main bronchus. Very soon after the chest wall had been opened and liberation of the left main bronchus began the man stopped breathing. We were called in to intubate him and observed the same phenomenon as heretofore. Under considerable pressure it was possible to inflate the lung but the lung remained distended. It was at this point that Prof. Lushnikov tried to compress the lung manually. The lung collapsed readily and was then re-inflated by us. This was done to see if for some reason or other a one-way valve had formed inside the bronchus. The idea of lung massage had not yet been thought of and no systematic massage was undertaken. Very soon the heart stopped. It was massaged assiduously with the same poor result as in the first case.

These two cases made us think that a total bronchospasm is an arrest of the lung similar to cardiac arrest and could perhaps, for want of better methods, be treated along similar lines. A paper by Muendnich[2] explained, we felt, the biochemical background of bronchospasm. The anæsthetist can inflate the lungs during bronchospasm and, therefore, during the first minutes oxygenation is still adequate and the oximetry readings satisfactory. But the elastic force of the alveolar walls is too insignificant to force the carbon dioxide-laden air through the spastic bronchiolar muscles and hypercapnia must necessarily develop. Hypercapnia means deepening of narcosis, hence the dilating pupils and the flat electroencephalographic curve. (In our second case under local analgesia no narcotic agent had been used). Hypercapnia attacks the myocardium next and as this begins to fail we see our oximetry curve go down. This is hypoxia of circulatory origin. Then the heart stops. It is therefore obvious that it is pointless to massage a heart which has stopped due to hypercapnia caused by pulmonary arrest. We would achieve nothing by chasing the toxic,

hypercapnic blood around. The lung massage must then precede that of the heart or else be undertaken simultaneously.

If diagnosis of total bronchospasm presents no special difficulties with the chest already open, it is less easy with the intact chest wall. One is naturally reluctant to slit the chest wall open every time the anæsthetist reports difficult ventilation.

However, there exist symptoms and signs which would make the diagnosis of bronchospasm so extremely probable as to justify surgical measures. Bronchial asthma or any other chronic disease of the lung in the patient's past history should serve as a warning to the anæsthetist. Thiobarbiturate used for induction must also be considered. Difficult ventilation which is not removed by addition of muscle relaxant and re-checking of the endotracheal tube for patency and correct positioning, total silence in the lungs upon auscultation during inflation – all these suggest total bronchospasm. Signs of hypercapnia (dilating pupils, flattening of the EEG despite no increase in the dosage of the narcotic agent), good oximetry figures at first, then their rapid decline. These are the signs of impending cardiac arrest.

One is tempted of course, to try various spasmolytic agents. Ideally it should be so and the answer to this problem must in the end be pharmacological and not surgical. Unfortunately total bronchospasm can apparently be provoked by a host of factors and we do not have a universal spasmolytic agent effective in every case. It is useless to inject one drug after another and wait for the bronchospasm to cease, as the minutes are much too precious.

Theoretically, massage of a single lung would suffice to treat hypercapnia, for we know that during thoracic operations one lung can take over the functions of the collapsed lung. This is especially obvious in lung operations done under local analgesia.

As our suggestion to massage the arrested lung manually was much criticised as being too traumatic (though it is much less so than cardiac massage), Dr N. F. Mistakopoulo[3] of our laboratory has constructed a very simple and efficient apparatus for air massage of one or both lungs and has successfully demonstrated on dogs that his apparatus will remove total bronchospasm provoked by a lethal dose of neostigmine. He intubates the trachea and both pleural cavities, through small incisions, and injects a lethal dose of neostigmine. Once the bronchospasm develops he inflates the lungs through the endotracheal tube, then compresses them by blowing air through the pleural tubes.

All the experimental evidence that he has so far collected, including the X-ray films of the bronchi filled with lipiodol at the height of bronchospasm and after its removal, the EEG and the ECG data and the spirometry readings are published in his paper[3]. Of his ten dogs

nine survived the lethal dose of neostigmine. (One was killed deliberately by injecting neostigmine and doing nothing further.) Another dog was successfully ventilated for some time, then the bronchospasm was removed by injection of atropine. But the remaining eight were saved only by air lung massage. Apparently if one ventilates the animal long enough the neostigmine level falls so much that it cannot maintain the bronchospasm. All animals were found to be perfectly healthy the next day. Mistakopoulo has also been able to show that all the abnormal changes in the blood pressure, ECG and EEG which develop following the appearance of a bronchospasm are removed once the air massage of the lung is started. He ventilated his dogs in this manner for a period ranging from 28 to 60 minutes, after which the bronchospasm completely ceased.

In December 1958 we had an occasion to use direct massage of the lung on a mitral case.

> A young man suffering from advanced mitral stenosis was given diplacin (a long-acting non-depolarising relaxant) first. We then proceeded with thiopentone. Before consciousness was lost he complained of difficult breathing (which we attributed to the action of relaxant) and tried to lift himself up. After intubation we found that inflation was impossible. Endotracheal tube was withdrawn and examined. The patient was re-intubated. Bronchospasm was suspected half-heartedly and atropine and ephedrine were given without effect. As oximetry figures began to fall and the pupils started to dilate to dangerous proportions, the lungs were auscultated. There was total silence. The surgeon was asked to open the chest and start lung massage. This was done and he was within the chest wall in two minutes. At this point the heart had stopped. The lung was inflated and would not collapse. Lung and heart were massaged simultaneously. The lung reacted to massage surprisingly quickly. Within a couple of minutes ventilation became entirely free and unimpeded. The size of the pupils reverted to normal very rapidly. The heart responded to massage too. Within six minutes after the chest wall was open the surgeon (Dr V. M. Buyanov) performed mitral commissurotomy. The heart started to fibrillate. Defibrillator was used successfully. The patient did not awake on the operating table for some time despite the low concentration of ether that had been used and cerebral damage due to hypoxia was suspected. This was indeed found to be the case when he finally awoke. He had paresis of the left arm and severe headaches for several weeks. He was finally discharged in good condition.

This is the first case to our knowledge when the surgeon opened the chest for the purpose of massaging the lung and we believe that it lends support both to the theoretical considerations and the experimental work of Dr Mistakopoulo. It is of course possible, that the bronchospasm in this case would have ceased spontaneously. This is however unlikely, as the extra two minutes of useless inflation of the lungs resulted in cardiac arrest.

SUMMARY

It is suggested that total bronchospasm of unknown ætiology can be treated by massaging the lung.

Apparatus for air massage of the lung and experimental work on neostigmine-induced bronchospasm in animals is described.

A description of a case of lung massage for total bronchospasm in man is given.

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This article is printed with but minor editorial corrections to the style. Readers will thus be able to appreciate Dr Smolnikoff's excellent command of colloquial English. (Editor, *Anæsthesia*).

Functional infundibular stenosis

treated by cyclopropane anæsthesia

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Indications for the use of general anæsthetics as therapeutic agents are relatively uncommon. A recent case in this hospital is therefore reported.

CASE HISTORY

A girl aged six months with Fallot's tetralogy was admitted with an acute respiratory infection. On examination, she was cyanosed and dyspnœic. The air entry to the right lung was impaired and there were scattered rhonchi. The liver was enlarged two finger-breadths below the costal margin. The child was placed in an oxygen tent and given antibiotics together with digoxin 0.1.mg twelve hourly by mouth, but deteriorated, becoming more cyanosed and distressed. She was seen by Dr R. V. Gibson who diagnosed acute infundibular stenosis[1] and advised therapeutic cyclopropane anæsthesia and withdrawal of the digoxin.

No premedication was given. The child was removed from the tent and given oxygen (4 litres/min) using a face mask and Ruben valve. The ECG recording was commenced (tracing A). Cyclopropane (300ml/min) was added to the oxygen. Difficulty was experienced because the mask did not fit well. The flow of cyclopropane was therefore increased to 750ml/min. During the course of the anæsthesia, the murmur became louder. After ten minutes, the ECG showed bradycardia (tracing B). The cyclopropane was stopped, the accumulated pharyngeal secretions sucked out and on inhaling

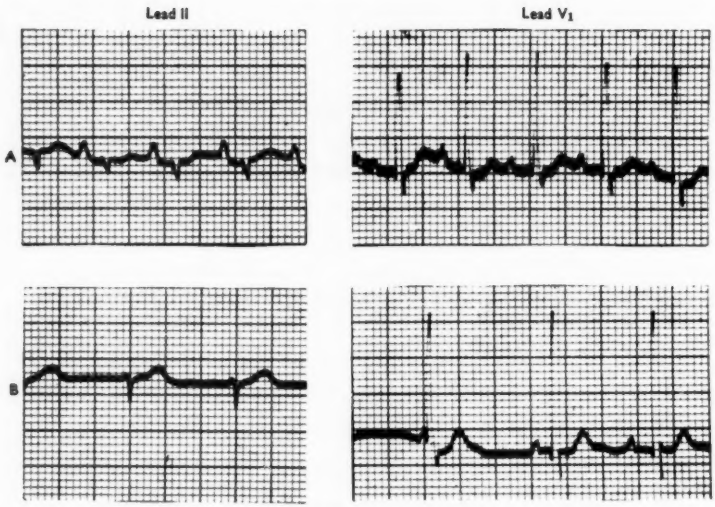

FIGURE TRACING A E.C.G. during preliminary oxygenation.
TRACING B E.C.G. after 10 minutes cyclopropane and oxygen
(Time-marker: heavy lines 0.2 second)

oxygen, the reflexes soon returned. The child was then replaced in the tent, the heart rate having risen to its previous level.

Next day she appeared somewhat better, but after forty-eight hours and following a feed, she became distressed, dyspnœic and more cyanosed. On examination, the trachea was deviated to the right and the air entry to the right lung diminished. A diagnosis of right bronchial obstruction was made.

Again cyclopropane anæsthesia was induced and bronchoscopy carried out using the suckling size Negus-type instrument. Mucopurulent material was found in both main bronchi and this was removed by suction. Again the murmur became louder and bradycardia developed. At the conclusion, active pharyngeal reflexes soon returned. On replacing the child in the tent, her respiration appeared easier and she seemed less distressed.

A continuous improvement in the child's condition occurred during the following month.

DISCUSSION

This use of cyclopropane is based on the observations of Wood[1]. He studied five cases of Fallot's tetralogy with a history of syncope and concluded that such attacks were due to functional obstruction in the infundibulum producing anoxia. During a cyanotic attack in the

course of catheterisation of one such case, the murmur shortened and the pulmonary artery pressure fell. There was little improvement with oxygen but on adding cyclopropane the cyanosis was relieved, the murmur became loud and long, and the pulmonary artery pressure increased to normal. He concluded that cyclopropane relieved the infundibular over-action, but doubted if this drug was specific.

In the child who is the subject of this report, the clinical picture was complicated by the chest infection and by bronchial obstruction. On both occasions cyclopropane was administered, the acute infundibular obstruction appeared to be relieved as shown by the increase in the intensity of the murmur. The beneficial effect of this form of therapy was shown by the immediate improvement in the child's condition.

The means by which cyclopropane relieves this functional stenosis is not definitely known. Li and Etsten[2] suggest that myocardial function is not impaired during surgical levels of cyclopropane anæsthesia in man, but that cardiac output is reduced and attributed this to the reduction in heart rate and to the rise in total peripheral resistance. It would seem difficult to explain the beneficial effect of cyclopropane on acute infundibular stenosis unless in fact it does have an effect similar to chloroform, depressing the myocardium, especially as at the time hypoxia may be present. Although the force of contraction would be reduced, the relief of the stenosis could more than compensate for this. Cyclopropane has however been shown[3] to increase the pressure in the pulmonary artery and this is thought to be due to a direct pressor effect on the pulmonary vascular bed. Moreover it would seem that the use of digoxin could make the situation worse by improving the strength of the heart's contraction, and it was therefore withdrawn in this case.

Braudo and Zion[4] confirmed Wood's hypothesis that these cyanotic attacks are precipitated by increased obstruction to the pulmonary outflow tract.

In view of these reports, the authors advocate that cyclopropane be available whenever cardiac catheterisation is undertaken.

SUMMARY

Functional infundibular stenosis in a child with Fallot's tetralogy was relieved by cyclopropane anæsthesia.

Acknowledgements

We would like to thank Dr P. N. Swift for permission to record this case and we are indebted to Dr R. V. Gibson for his advice.

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Drugs which stimulate affective behaviour

1 Action of lysergic acid diethylamide (LSD-25) against thiopentone anæsthesia in dogs

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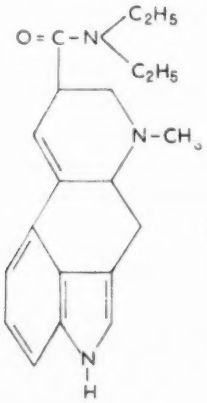
Several compounds which are under clinical and experimental trial have the peculiar property of stimulating affective behaviour in a way that is somewhat different to caffeine and amphetamine, but seem to have an alerting effect also[1]. Some of these drugs may prove to be of great value to the anæsthetist for reversing barbiturate anæsthesia or poisoning.

One of these drugs is delysid (lysergic acid diethylamide or LSD-25). This compound is a semi-synthetic derivative obtained by the condensation of d-lysergic acid (an extract of ergot) with diethylamine (a secondary amine). It is one of the ergonovine group of drugs and was first isolated by Stoll and Hofmann in 1938[2]. The structural formula is:



L S D - 25
(delysid)

In 1943 Hofmann observed that LSD-25 has an unusual psychic effect in man[3]. This compound is now being used mainly to produce model psychoses for the clinical investigation of the mechanism and biochemical basis for schizophrenia[4].

A number of investigators reported that LSD-25 appears to antagonise the action of anæsthetic doses of barbiturates, on the basis of electrophysiological evidence in rats, rabbits and cats. LSD-25, like amphetamine, produces alerting behaviour and abolishes the EEG spindle activity of barbiturates in cats[5 6 7]. Apter carried out extensive studies with cats, and showed that LSD-25 was more effective than picrotoxin or pentamethylenetetrazol (metrazol) in preventing death from a lethal dose of pentobarbitone and was also more effective than picrotoxin in reversing the cortical suppression and cessation of respiration induced by a previously administered lethal dose of pentobarbitone[8]. The dose of LSD-25 solution necessary to prevent and to reverse pentobarbitone death was harmless to vital functions in both unanæsthetised and anæsthetised cats, whereas the dose of picrotoxin necessary to reverse pentobarbitone toxicity was convulsive or subconvulsive in anæsthetised cats.

The results of these studies indicated that LSD-25 might have a place in the therapy of barbiturate poisoning in humans. The following studies were undertaken to determine whether LSD-25 has a demonstrable analeptic effect on dogs under anæsthesia with thiopentone.

METHOD

Acute experiments were carried out first in six dogs as follows. Each animal was lightly anæsthetised intravenously with a 'sleep dose' of thiopentone. A solution of 5 per cent dextrose in water was attached to the intravenous needle to assure adequate hydration and to keep the vein open. A cuffed endotracheal tube was placed in the trachea immediately following induction of anæsthesia. This was attached to a positive-negative pressure respirator and to a tank of oxygen, to assure adequate pulmonary ventilation throughout the study. Cycling rate of the respirator was adjusted at 30 to 35/minute and the minute volume of ventilation was set between 3 to 4 litres of oxygen. The electrocardiogram (lead 2), electroencephalogram (fronto-occipital leads), pneumotachygram and arterial blood pressure from the abdominal aorta (cannula threaded into femoral artery) were recorded simultaneously on a Grass-direct writing polygraph. The recordings were continuous throughout each experiment. After control recordings were taken, an estimated lethal dose of thiopentone was injected rapidly (75 to 125mg/kg). In each animal this dose was greatly in excess of the dose which would cause lethal apnœa

if respiration were not controlled[9]. When cardiovascular depression was evident from a severe fall in aortic blood pressure, LSD-25 was administered in a dose of 50 to 100μg/kg. In each experiment artificial respiration was provided until the animal became responsive or expired.

Cross-over experiments were carried out then on ten mongrel dogs of comparable size and age at four weekly intervals. The method employed was similar to that reported by Virtue and Kaster[10]. Eight of these dogs were from the same litter. Thiopentone was administered in a single intravenous dose to each dog. The dose was fixed at 25mg/kg. The thiopentone was injected at the approximate rate of 200mg/minute in a 2 per cent solution. At each experiment alternate groups of five dogs were given 25μg/kg of LSD-25 intravenously one minute after the injection of thiopentone. After the injections, the dogs were placed on the floor of the laboratory and allowed to recover without being disturbed or stimulated. The recovery time of each animal was recorded as judged by the time which elapsed from completing the injection until the head was raised spontaneously and until the dog was able to stand on all four paws without collapsing. As soon as a dog recovered, it was removed from the room so that it would not stimulate the other animals.

RESULTS

Data from the acute experiments are shown in TABLE 1. After the injection of LSD-25, there was no obvious effect on the recorded vital

Table 1

Acute tests of the analeptic effect of LSD-25 on a lethal dose of thiopental in dogs

DOG	WEIGHT KG	DOSE THIOPENTAL MG	DOSE LSD-25 μG	DURATION OF APNŒA MIN	SUPPORTIVE DRUGS
1	9.0	1350	550	Survival SR 140	phenylephrine
2	7.0	1050	350	Died	phenylephrine epinephrine nikethimide
3	8.0	825	480	Survival S.R. 168	—
4	5.5	400	275	Survival SR 82	—
5	6.0	550	350	Survival SR 97	—
6	5.5	425	300	Survival SR 118	—

SR = Spontaneous Respiration

signs for several minutes. Then there was some increase in cortical activity on the EEG in dog numbers 1, 3, 4 and 6, but no change in the electrocardiogram, arterial blood pressure or breathing pattern.

One dog expired. The electrical voltage of the EEG fell during a period of forty minutes and the arterial blood pressure was unrecordable from the aorta within twelve minutes of the injection of the thiopentone. The subsequent administration of neosynephrine, epinephrine and coramine did not improve the condition of this dog and it was obviously dead after resuscitation efforts had been continued for three hours.

After a period of 80 to 170 minutes, respiratory efforts became evident in the rest of the dogs and there was a gradual return of the arterial blood pressure. When the respirator was disconnected, there was slow but progressive recovery of respiration and wakefulness. Dogs 1 and 3 did not rise and take food until the day following the experiment.

Although it appeared as if LSD-25 may have accelerated the recovery of the surviving dogs, the evidence was not unequivocal because artificial respiration with oxygen alone often sees the animal through the lethal effects of barbiturates, as is observed in humans who have been poisoned by a very excessive dose of barbiturates. Since it is difficult to carry out reliable and valid experiments of this nature without a large population of dogs and without a known consistent range of lethal doses of thiopentone, it was decided to abandon this line of tests in favour of the cross-over type of experiment.

A summary of the data from the cross-over experiments appear in TABLE 2. The apnœa which followed the rapid injection of thiopentone was not always reversed immediately by LSD-25, as one usually sees with an analeptic drug. There was no significant difference in the recovery time between the experiments with or without LSD-25. Although it appeared as if the animals that received LSD-25 were more alert and were able to walk about a little sooner, the Fisher t test applied to these data showed that the difference was not statistically significant.

DISCUSSION

In the unanæsthetised dog, when LSD-25 is injected directly into the third cerebral ventricle in doses of up to 10μg/kg, the animal reacts within a minute or two in the following sequence: whining and shaking of the head, licking the chops, salivation and ataxia; retching, nausea, emesis, micturition and tachypnea. After five minutes the reaction is exaggerated, and mydriasis with reactive pupils are observed. Recovery occurs after 15 to 20 minutes. Throughout the

Table 2

Test of the analeptic action of LSD-25 on thiopentone narcosis in dogs

	WEIGHT	FIRST WEEK		SECOND WEEK		THIRD WEEK		FOURTH WEEK	
		RECOVERY TIME HEAD UP LEGS UP		RECOVERY TIME HEAD UP LEGS UP		RECOVERY TIME HEAD UP LEGS UP		RECOVERY TIME HEAD UP LEGS UP	
DOG	KG	DRUG	MIN	DRUG	MIN	DRUG	MIN	DRUG	MIN
1	9.5	Thiopental	47	Thiopental	66	Thiopental	36	Thiopental	64
		LSD-25	110	LSD-25	85	LSD-25	58	LSD-25	73
2	8.6	Thiopental	75	Thiopental	82	Thiopental	54	Thiopental	72
		LSD-25	145	LSD-25	91	LSD-25	65	LSD-25	86
3	12.3	Thiopental	34	Thiopental	0	Thiopental	33	Thiopental	49
		LSD-25	46	LSD-25	0	LSD-25	36	LSD-25	55
4	9.5	Thiopental	26	Thiopental	84	Thiopental	58	Thiopental	49
		LSD-25	45	LSD-25	97	LSD-25	68	LSD-25	57
5	9.1	Thiopental	36	Thiopental	94	Thiopental	64	Thiopental	43
		LSD-25	60	LSD-25	148	LSD-25	70	LSD-25	54
6	11.8	Thiopental	58	Thiopental	118	Thiopental	63	Thiopental	35
		LSD-25	71	LSD-25	216	LSD-25	84	LSD-25	42
7	10.9	Thiopental	50	Thiopental	67	Thiopental	51	Thiopental	54
		LSD-25	67	LSD-25	73	LSD-25	65	LSD-25	60
8	13.6	Thiopental	4	Thiopental	55	Thiopental	36	Thiopental	28
		LSD-25	15	LSD-25	67	LSD-25	39	LSD-25	37
9	13.2	Thiopental	51	Thiopental	26	Thiopental	35	Thiopental	62
		LSD-25	57	LSD-25	41	LSD-25	39	LSD-25	78
10	14.5	Thiopental	39	Thiopental	63	Thiopental	51	Thiopental	56
		LSD-25	47	LSD-25	76	LSD-25	71	LSD-25	69

Average recovery time — Thiopental alone (20 administrations): Head up: 51 minutes (A) Legs up: 73 minutes (B)

Average recovery time — Thiopental + LSD-25 (20 administrations): Head up: 52 minutes (C) Legs up : 65 minutes (D)

Critical ratio (Fisher t test), A – C = 0.2, B – D = 0.6

period after the injection, the animals appear frightened but there is no impairment of their ability to follow simple commands or perform simple tasks. Similar responses were observed with larger

doses of LSD-25 when injected intravenously[11]. Others have also reported the stimulating effect of LSD-25 on sympathetic and reflex activity[12 13]. These observations point to a possible analeptic-type of action against barbiturate intoxication.

The influence of LSD-25 on survival after a lethal dose of thiopentone is very difficult to demonstrate reliably because the difference between the dose which produces respiratory arrest and fatal cardiovascular collapse varies greatly, even when all the experimental conditions such as the rate of injection and concentration are controlled. This variation is so wide among dogs that it is not feasible to carry out a well-designed experiment which might demonstrate with certainty the drug protection against a lethal dose of a barbiturate.

The cross-over experiments demonstrated more simply whether the drug was effective in shortening the recovery time from thiopentone anæsthesia. No significant analeptic effect was observed in these experiments. However, the effect of LSD-25 may be such that it cannot be shown objectively, and it is unfortunately no simple task to design an experiment for obtaining suitable subjective data from dogs.

SUMMARY

Acute and cross-over experiments were employed to determine whether LSD-25 has an analeptic effect against thiopentone anæsthesia in dogs. In the acute experiments, a lethal dose of thiopentone was administered, followed by LSD-25. Artificial respiration with oxygen was provided with a mechanical automatic positive-negative pressure respirator until recovery or death was obvious. Although only one of six dogs died, the evidence of an analeptic action by LSD-25 was not unequivocal. In the cross-over experiments, ten dogs were each given thiopentone alone and thiopentone followed by LSD-25 alternatively at four weekly intervals. The short period of apnœa which follows the rapid injection of thiopentone was not always reversed immediately by the injection of LSD-25, and there was no statistically significant analeptic effect with LSD-25 as determined by the time interval for the dogs to raise the head and to rise on their paws without collapsing.

Acknowledgements

Mr Stan Meakin and Mr B. St. Germaine of the Medical Research Laboratory, University of Saskatchewan, provided very helpful technical assistance during these experiments. Statistical analysis of the data was carried out by Prof. N. Shklov, from the Department of Mathematics, University of Saskatchewan. The authors are grateful to J. M. Grosheintz, D.SC., Director of Medical Department of Sandoz Ltd, Montreal, Canada, who supplied the d'lysergic acid diethylamide tartrate (delysid, LSD-25).

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VOL 15 NO 1 ANÆSTHESIA JANUARY 1960

Effect of decamethonium upon conditioned reflexes in rats

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Investigation of the methonium compounds by Paton and Zaimis[1] showed that decamethonium (C_{10}) possessed a powerful neuromuscular blocking action. Following this report decamethonium was introduced in clinical practice where it found a place as a depolarising muscle relaxant with a moderately short duration of action[2 3]. Although this drug has never been used so extensively as curare in clinical anæsthesia, it has remained a favourite means of inducing muscular paralysis in experimental procedures. It was whilst using decamethonium in experimental animals that Waller and Smith[4] noticed changes in the response of animals to acquired reflexes. They showed that animals partially paralysed by this drug reacted abnormally in response to certain acquired visual reflexes. The work presented in this paper is the result of a preliminary investigation performed to test the validity of their findings and to try to elucidate the mechanism of this particular effect of decamethonium.

METHOD

Twelve white rats of the same stock and of approximately the same age and weight were selected for the initial experiments. They were divided into four groups of three. One group was trained to respond to a visual conditioned reflex, the second to an auditory conditioned reflex, and the third group to tactile stimuli. Three rats were retained to act as controls to study the extent of the paralysis induced by the dose range of methonium used in the experiments. Unfortunately one of the control animals died and one in the group conditioned to respond to visual stimuli, had to be withdrawn from the study because of pregnancy.

The rats were considered fully trained when they showed consistently for two days a 95 per cent correct response during the thirty

This investigation was carried out whilst the author was in receipt of a Research Fellowship in Anesthesiology at the University of Washington, U.S.A.

minute test period. When they were suitably trained a subparalytic dose of decamethonium iodide was injected intraperitoneally and the response to the conditioned reflexes was again tested. In all these experiments two dose levels of decamethonium were used; 0.4mg/kg and 0.8mg/kg. These were found to be the minimal effective doses when administered intraperitoneally, but they are far below the intravenous dose of 2.5mg/kg which is accepted as necessary to produce paralysis in rats[1]. To avoid the possibility of tachyphylaxis not more than one injection of the drug was given to any animal in one day. Each animal acted as its own control, an intraperitoneal injection of saline being given in place of the active drug.

TRAINING

A simple cage was constructed, consisting of two compartments separated by a partition with a communicating door. The floor of each compartment was covered with copper bars wired to form a grid, so that contact between any two consecutive bars would complete an electrical circuit. The floor of each compartment could be electrified separately and an animal in the electrified compartment would receive a weak shock which would encourage it to run through the communicating door into the 'safe' compartment.

Two rats were trained to respond to a visual conditioned reflex by flashing a light in the roof of the compartment four to five seconds before the grid was electrified. They soon became trained to leave the lighted compartment in order to avoid the electric shock. Three rats were similarly trained to change compartments following a repetitive tap on the roof of the cage. The third set of rats were conditioned to move from one compartment to another upon receiving a gentle blast of air of sufficient intensity to ruffle the fur upon their backs. Each rat received individual training for a period of approximately thirty minutes each day, during which time the conditioning procedure was repeated every ten to fifteen seconds.

The untrained rats reacted to the electric shock by purposeless rage, squealing and jumping up at the side walls of the cage. Later when they were partially trained, if they ignored the conditioning stimulus, their response to the electric shock was purposeful and they would seek the 'safe' compartment.

The control rats were used to test the reduction in spontaneous activity induced by an intraperitoneal injection of 0.4mg/kg and 0.8mg/kg of decamethonium, as compared with the effect of an intraperitoneal injection of saline. For this purpose a 'jiggle cage' was used. This is a delicately balanced cage with a counting mechanism that computes the number of movements made by the rat that were

of sufficient intensity to trip the counting mechanism. The rat under test was allowed thirty minutes to become used to the cage and was then given either an injection of saline or of decamethonium and the number of movements in the following thirty minutes were recorded. The control rats were tested each day with decamethonium and with saline. Each rat was tested twice at both dose levels of the drug.

RESULTS

The three groups of rats showed marked difference in the length of time required to attain 95 per cent proficiency in response to the conditioning stimuli. The rats in each group were fairly consistent in

Table 1

No. of days training required to attain 95 per cent correct response

	NO. OF DAYS TRAINING
Visual conditioned reflex	9
Auditory conditioned reflex	11
Tactile conditioned reflex	8

the number of days training required, but a little extra time each day was spent with the more backward rats in order to simplify the testing procedure.

The result of the administration of decamethonium upon the rats' response to conditioned stimuli is illustrated in FIG. 1. The number of correct responses to the conditioning stimulus was recorded during each five minute period and expressed as a mean percentage of correct performance for the two or three rats tested at a particular dose level. Most animals showed no apparent objective change following the intraperitoneal injection of either 0.4mg/kg or 0.8mg/kg of decamethonium. They could stand, walk and jump normally. Occasionally muscular fasiculation was seen following the administration of 0.8mg/kg of the drug.

The response to auditory stimuli, which necessitated the longest period of training, proved the most affected by decamethonium. Following 0.4mg/kg of the drug no response could be elicited for twenty minutes, which is consistent with the duration of action of the drug. The visual conditioned reflex was also completely depressed after a dose of 0.4mg/kg of decamethonium, but for a shorter period. Tactile conditioned reflexes, which had been the easiest to establish proved most resistant to decamethonium, 0.8mg/kg of the drug being required to abolish the animal's ability to respond correctly. The control injection of saline produced a small temporary diminution in the animal's performance, an effect that was approximately equal in

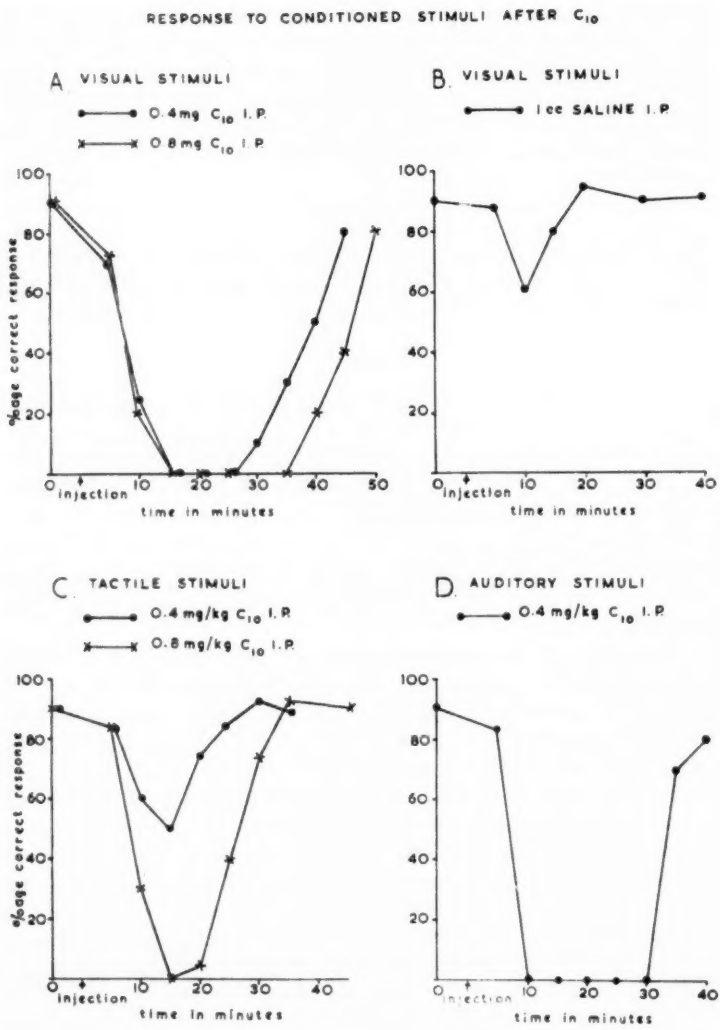

FIG. 1

all the groups. The results of the injection of saline in the group conditioned to respond to visual stimuli are included in FIG. 1.

During the period that the rats were under the influence of the drug and failing to respond to the conditioning stimulus, their responses to the grid shock were also modified. They no longer sought out the

'safe' compartment but behaved like untrained rats, jumping and squealing in a purposeless manner. As the effect of the drug wore off the rats rapidly regained the ability to respond to the conditioned reflex.

Testing the effect of decamethonium in the dosages used upon the control rats in a 'jiggle' cage, revealed no demonstrable depression of spontaneous activity after an intraperitoneal injection of 0.4mg/kg as compared with an injection of saline. An average of 7 per cent decrease in spontaneous activity was recorded following the administration of 0.8mg/kg of the drug. This confirmed the impression that the degree of muscle paralysis induced by this dose of decamethonium was very small and certainly by itself insufficient to account for the loss of response to conditioned reflexes.

DISCUSSION AND SUMMARY

It appears from the results of the experiments described, that decamethonium administered in sub-paralytic doses, abolished certain acquired conditioned reflexes in rats. The duration of this effect coincides with the expected duration of action of the drug.

The interpretation of these results presents certain problems if one accepts the view that the action of decamethonium is localised to the neuromuscular junction.

It has been known since Pavlov's classic experiments on conditioned reflexes, that animals may lose their response to conditioned reflexes if subjected to a particularly fearful circumstance. The injection of a small dose of decamethonium did not appear to be a particularly traumatic experience and the rats showed no sign of fear or rage. Unlike Pavlov's dogs, the rats subjected to this experiment did not lose their ability to respond permanently but only for the duration of action of the drug. It is probable that some other mechanism was responsible for the failure of the rats to respond after the injection of decamethonium and that the drug had effected some alteration in the central nervous system.

Many experiments have been performed in order to study the possible central nervous system action of muscle relaxants. The evidence for the existence of such an action is mainly indirect. Changes have been described in the EEG of animals subjected to the injection of curare by various routes[5-10]. Prolonged respiratory depression occasionally seen following the use of curare has been attributed to an action of the drug upon the central nervous system[11], this theory having received some experimental support[12]. Direct observations following the administration of curare[13], decamethonium[2], and suxamethonium[14] to conscious volunteers, do not support the suggested action upon the central nervous system.

An interesting observation was made by Girden and Culler[15] who found that dogs conditioned to respond to an auditory stimulus whilst partially paralysed with curare, would not respond to the same stimulus in the absence of curare.

It is difficult to envisage an action of the muscle relaxants upon the central nervous system that would suitably explain both the observations of Girden and Culler with curare, and those reported in this paper with decamethonium and yet be reconcilable with the assertion of volunteers who have received these drugs, that their mental acuity remained unaltered. A further objection to any theory interpreting these results as a direct action of the drug upon the brain is the small amount of drug used. McIntyre *et al*[16] found that only if large doses of muscle relaxants were given could it be expected that they would cross the blood brain barrier. It seems very unlikely that this would be achieved with the small doses of relaxant used in these experiments.

An alternative theory has been proposed by Paton[17]. He suggested that, as a result of the blockade by relaxants of the synapses between the γ nerve fibres and the muscle spindles, the normal afferent passage of stimulus from the muscle receptors to the brain is reduced and (apparent) changes in the central nervous system may result. Thus, as a result of their peripheral actions, muscle relaxants may alter the modality of sensory appreciation by the brain. This theory would suitably explain the findings reported in this paper and those of Girden and Culler, as the central interpretation of the afferent sensory stimuli from the special sensory receptors may have been modified by an alteration in the normal neural background activity consequent to a change in the afferent discharge from the stretch receptors.

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The resistance of expiratory valves in actual use

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The characteristics of expiratory valves have been investigated by Mushin and Mapleson[1]. Their work however was done under static conditions with continuous gas flow. Respiration on the other hand involves to and fro movement of air with rapid changes in velocity. It is therefore possible that the findings of experiments done with continuous flows are not directly transferable to clinical anæsthesia, just as the findings concerning the resistance of an electric circuit to direct current are not applicable where the current is alternating.

The compressible nature of gases, indeed, makes it almost certain that the resistance opposed by a valve to an intermittent flow will be different from that to a continuous flow. For the first result of the developing obstruction to the flow produced by a valve must be a compression of the gases. In other words the elastic nature of gases has an initial cushioning effect on the pressure which is built up behind an obstruction. Similarly when the period of obstruction comes to an end there must be a short delay in the disappearance of this pressure while the compression of the gases reduces itself. The general result is rounding out of sharp angles in the pressure time curve with a cutting off of its peaks and perhaps a slight prolongation of the effects of a single disturbance. For these reasons it was felt that the work hitherto done with continuous flows of gas was not perhaps directly applicable to the changes which occur during clinical anæsthesia. Observations were therefore made on actual anæsthetised patients.

METHODS

The investigations were carried out at the end of major chest operations under nitrous oxide-oxygen anæsthesia when the valve was already damp from condensed moisture. Either Carlens tubes or ordinary cuffed endotracheal tubes were in situ. During the operations the patients had been ventilated with an Aintree respirator and the bag and elephant tubing of this apparatus were retained during the

studies. Where semi-closed anæsthesia was required the apparatus was used with the Heidbrink expiratory valve fully open, close to the patient and upright with the bag at the far end of the unkinkable hose. When it was necessary to cut out the expiratory valve the hose leading to the bag was disconnected and opened to the atmosphere at the junction midway along its length.

Pressure differences were recorded with the aid of a Southern Instruments Capacitance Manometer. Where intra-œsophageal pressure changes were determined, a manometer head was connected to a semi-rigid gum elastic catheter passed into the œsophagus. A very slow (5–10 drops/min) flow of water through the catheter was employed to ensure its continued patency.

Pressure changes on the gaseous side were determined either by connecting a manometer head, with the aid of a short length of rubber tubing, directly to the suction end of a Cobb connection or to one limb of a glass Y-junction let into the tube feeding anæsthetic gases to the patient and immediately adjacent to the expiratory valve. In three cases the airway pressure changes at bifurcation of the trachea were measured instead. In these cases a very fine gum elastic suction catheter was passed beyond the open end of the endotracheal tube, an airtight fit between the suction end of the Cobb connection was produced and the catheter was filled with water to ensure that the pressure changes were recorded without distortion.

RESULTS

The amplitude of the pressure swing produced by normal breathing was measured in nine patients with and without an expiratory valve. When the valve was in use the mean respiratory pressure swing was 3.9 ± 1.5cm of water. When the expiratory valve had been removed and the same patients were breathing freely in and out of an open elephant tube the pressure swing associated with respiration was 3.2 ± 1.4cm of water. The differences in these two figures is very little more than its standard deviation and is not statistically significant. The pressure which was measured in the experiment was however the sum of all the resistances to respiratory gas flow. It was not wholly due to the expiratory valve and included a component due to the Cobb connection.

The form of the pressure wave with the two forms of anæsthesia was next recorded, FIG. 1. When the expiratory valve was in use the onset of inspiration was marked by a sharp fall in pressure with a return to a resting level which remained more or less constant not merely to the end of expiration but until the next inspiration began. With open circuit anæsthesia there was a typical biphasic curve with

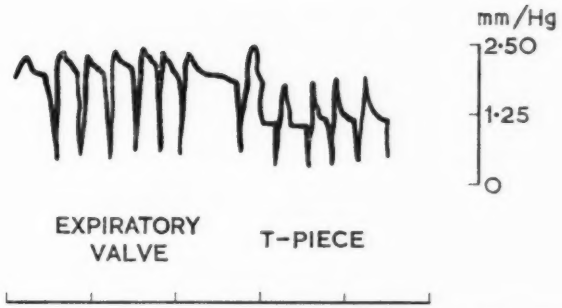

FIG. 1 The pressure wave forms associated with the use of an expiratory valve and T-piece anæsthesia. (Time marker: 10 seconds).

equal negative and positive inspiratory and expiratory pressure swings and a resting level midway between. The difference in this resting pressure was measured in twelve cases and found to be 2.0 ± 0.99cm water. The result of this difference was that the mean pressure in the airway, as measured in three cases, was between 2 to 3cm of water higher when the expiratory valve was used, than it was in its absence.

The extent to which these pressure changes were transmitted to the mediastinum was measured in the œsophagus. The pattern of the respiratory pressure wave was the same whether an expiratory valve was in use or not but removal of the valve caused a decline in the intra-œsophageal resting pressure of 1.2 ± 0.74cm water in twelve patients.

DISCUSSION

This investigation has shown that an expiratory valve which is clinically satisfactory does in fact oppose some resistance to the escape of gases through it. Its extent is very much what would have been expected from the static studies with continuous gas flows made by Mushin and Mapleson[1].

The airway pressure remains higher for a longer period of the respiratory cycle with an expiratory valve than it does with open circuit anæsthesia. This rise in airway pressure is transmitted to the œsophagus and presumably also to the great veins in the mediastinum where it must cause some damming back of the blood returning to the heart. The extent of the pressure change in the œsophagus is small, being of the order of only just over 1cm of water for the valves studied. This is what would have been expected from the author's previous work on the transmission of pressures to the œsophagus[2].

A rise of pressure in the œsophagus of 1cm of water is negligible compared with the pressure rises which occur for other reasons, *e.g.* in straining. It must therefore be doubtful whether the apparent good results obtained when T-piece anæsthesia is employed can be explained on the basis of the relative absence of obstruction to the venous return which prevails when this technique of anæsthesia is employed.

Acknowledgements

My thanks are due to the South Manchester Hospital Management Committee for a grant to cover the expenses of this investigation including the purchase of the capacitance manometer.

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SUMMARIES OF PAPERS

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ASSOCIATION OF ANÆSTHETISTS

STRATFORD-UPON-AVON

FRIDAY 23 OCTOBER 1959

Ventilation nomograms during anæsthesia

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Radford's nomogram has been reviewed with regard to its applicability under the special conditions of anæsthesia. Under these circumstances the carbon dioxide output would appear to be over-estimated and the dead space under-estimated. These errors being opposite in effect and approximately equal may be expected to cancel one another at normal tidal volumes.

In twelve patients ventilated artificially with the chest closed it was found that Radford's nomogram did, in fact, give an accurate guide to correct ventilation with little individual scatter. In the same patients during spontaneous respiration and in a further twelve patients during thoracotomy, the correlation between P_{CO_2} and ventilation was poor.

To avoid the errors inherent in Radford's approach, two new charts are presented for the prediction of P_{CO_2} from ventilation. The first of these is used to predict the carbon dioxide output of an anæsthetised patient from age, sex and surface area. The second chart relates P_{CO_2} to minute volume for a given carbon dioxide output (obtained from the first chart). As with Radford's nomogram, these new charts gave reasonably satisfactory results during artificial ventilation with the chest closed. However, during spontaneous respiration and during thoracotomy individual scatter made the predictions almost valueless although the mean error of prediction of the group was small.

It is thought that the scatter during spontaneous respiration was due to failure to attain a steady state, while during thoracotomy considerable variations in the physiological dead space occurred. Therefore, under these circumstances, the use of ventilation nomograms is of little value.

Changes in respiratory physiology of significance to anæsthesiologists

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With the advances in anæsthesia and surgery, operations are now offered patients who manifest increasingly complex derangements of physiology. Among these are the patients with severe chronic respiratory disease, and it becomes incumbent upon the anæsthetist that he be an astute student of pulmonary physiology. It becomes necessary not only to carry out a good pre-operative evaluation of the respiratory status of the patient, but it is also necessary that studies be done post-operatively and treatment be directed as indicated. An easily applicable method for evaluating pulmonary ventilatory function with a single-breath test is presented. With this one-breath test it is possible to determine with considerable accuracy the three important parts of pulmonary ventilatory function: volume displacement, velocity of air flow and intrapulmonary distribution. From this test a quadratic plotting system may be set up to show the limits of ventilatory function, categorising the patient as having normal, restrictive, obstructive, or a combined restrictive-obstructive defect in ventilation. With this information the anæsthetist can direct attention to the underlying difficulty by the use of intermittent positive pressure breathing procedures, nebulisation with bronchodilator-wetting agents, coughing exercises conducted with a special apparatus even for the non-co-operative patient, and aspiration procedures.

Ventilation during anæsthesia

R. WOOLMER, LONDON

The anæsthetist should aim to keep his patient's ventilation at a level which will maintain the arterial blood at a pH of 7.4 and a P_{CO_2} of 40mm Hg. Indirect methods of monitoring Pa_{CO_2} are described. As an alternative to monitoring P_{CO_2}, the rate of ventilation may be measured and adjusted to an ideal figure obtained by calculation. Methods of measuring the rate of ventilation are reviewed. A new Automatic Minute Volume Recorder is described. Records made

with this instrument show that the unaided anæsthetist, in spite of years of experience, is quite unreliable as a ventilating machine. The consequences of departure from normal CO_2 levels are briefly discussed and it is recommended that anæsthetists should attempt to avoid such departures.

The early history of ether

F. CARTWRIGHT, LONDON

Sulphuric ether is manufactured by the action of sulphuric acid upon alcohol; it is, therefore, possible to discard much of the legendary history of ether by fixing the dates of the first preparation of these two important chemical substances.

Sulphuric acid was first described and probably first prepared by the Arabian alchemist, Geber, about the year 760. The first distillation of spirits of wine is more certain for there is no doubt that this was the work of Raymond Lully in the thirteenth century. Lully is sometimes credited with the discovery of ether, as is Paracelsus and his pupil Valerius Cordus. From the description given by Paracelsus of his 'sweet oil of vitriol', there is little doubt that this was not ether, although it may have been a mixture of ether, alcohol and water. This preparation became famous as 'Hoffman's drops' and was classed as an anodyne drug.

Certainty comes with Wilhelm Godefroy Froben, a German chemist, who described the preparation of ether and gave the drug its present name in 1735. The name derives from a Greek word meaning 'to burn brilliantly' which was applied to the zenith, the quintessence of light and thence, by analogy, to the impalpable matter that separates one constellation from another. Thus, the term 'ether' has much the same significance as 'alcohol', as impalpable matter. Although ether became of some importance in medicine, it was in the sciences of chemistry and physics that its supreme usefulness lay, for it was the most volatile of all known fluids and therefore, particularly convenient for the purpose of studying the phenomena attending the change from the liquid to the gaseous phase. Lavoisier, during the course of experiments upon this subject, noted that, as ether boiled at about body temperature, it could only exist in the body as a vapour.

William Cullen of Edinburgh was the first to note (1773) that ether was only effective when vaporised. His description of the treament of headache may imply inhalation, but it was not until the 1790's that the

inhalation of ether came into popular practice with the vogue for pneumatic medicine. Richard Pearson of Birmingham claims, in a letter of 1795 to Thomas Beddoes, to have originated this form of treatment which he used for catarrh, croup and phthisis. It was also advocated by Thornton, Erasmus Darwin and John Collins Warren. The drug was now classed as an antispasmodic and was frequently used for asthma, a treatment which persisted into the twentieth century and is noted in a famous lecture by Sir Thomas Watson.

In 1800, as a result of Humphry Davy's writings, nitrous oxide became a scientific plaything. In 1818, Michael Faraday drew attention to the similarity between the action of nitrous oxide and ether and the latter, far more readily obtainable than nitrous oxide, became popular for its intoxicating action. In Scotland ether-drinking became prevalent after the closure of a number of the smaller distilleries; in England ether was used to adulterate gin, and gave rise to a disease known as and famous as 'the blues', which is noticed by Hart as an end result of habitual ether intoxication; in Russia, ether-drinking followed the prohibition of the sale of vodka by Imperial ukase in the 1914 war; the worst and most prolonged outbreak was in Ireland, following the siezure of numberless poteen stills. Here, in 1889, over five thousand pounds of ether were distributed in one small area comprising five villages. In the following year the Lord-Lieutenant forbad the sale of ether.

In America the 'ether frolic' never became widely popular and, outside academic circles, was practically confined to the Southern States, where the inhabitants were in the habit of distilling their own spirits at a strength approximating to that of absolute alcohol. It was these American 'frolics', however, which gave us the first attempts at surgical anæsthesia, those of William Clarke and Crawford Long in 1842 and of Smilie in 1844. None of these 'experiments' had any influence upon the introduction of anæsthesia, for the ether was used simply as a substitute for alcohol.

An entirely different approach was that of Horace Wells in 1844, using Humphry Davy's nitrous oxide. It was this experiment, which was dissimilar from ordinary intoxication, that fired Morton's flame of enthusiasm. The story of Morton's experiments is so confused that the exact truth will never be known. It seems certain that Morton used nitrous oxide, failed, and approached his teacher, Charles Jackson, for some further information concerning the gas. Jackson drew his attention to the similarity between the action of ether and nitrous oxide. Morton then tried ether, but he used the so-called 'commercial ether' which was distributed for its intoxicating effects; this was a weak and impure product obtained from adulterated spirit. Having failed again, Morton once more approached Jackson who

suggested the use of pure rectified ether. With this drug, still far from the standard of purity that we demand to-day, Morton, in September 1846, succeeded in inducing anæsthesia in the case of Eben Frost. Just over a fortnight later, on 16 October 1846, he gave a successful demonstration at the Massachusetts General Hospital. Thus ether came to the zenith of its strange history and, as the audience left the operating theatre, that surprising culmination was expressed in the words of Jacob Bigelow: *We have seen something to-day that will go round the whole world.*

Is it safe to employ hypotensive anæsthesia in the elderly ?

W. N. ROLLASON

J. M. HOUGH

ABERDEEN

The risks of hypotensive anæsthesia in the elderly have often been stressed due to the liability of thrombus formation and the danger of ischæmia due to atherosclerosis. The effect of a reduction in blood pressure on the oxygenation of the vital organs has been examined in the literature with a general conclusion that in healthy organs ischæmia is unlikely at systolic blood pressures of 60mm Hg or above.

Hypertension and atherosclerosis are common in the elderly and it might be thought that they would invalidate the above conclusion. However, it has been shown that advanced atheroma may even be a protection against clot formation and it is becoming normal practice to treat hypertensive patients with hypotensive drugs for long periods without serious consequences. In fact, the literature suggests that in the majority of cases hypotensive anæsthesia is likely to be beneficial to the circulation of the elderly.

A series of forty retropubic prostatectomies under hypotensive anæsthesia has been studied and compared with a larger series of cases done under normotensive anæsthesia. The morbidity and mortality was no worse under hypotensive anæsthesia. Electrocardiographic changes were studied for the hypotensive series and it was found that these became more serious when the systolic BP fell below 60mm Hg and that systolic hypertension seemed productive of these changes.

It is concluded that hypotensive anæsthesia is not absolutely contra-indicated for the elderly, even in cases of known coronary or cerebral incidents, provided the systolic blood pressure is not lowered below 60mm Hg, the anæsthetist is skilled and surgical advantages ensue from the technique.

Anæsthetic methods in animals

BARBARA WEAVER, BRISTOL

During surgical operations, animals must not, in accordance with the law, feel pain. Unlike humans, animals may be anæsthetised for restraint, *e.g.* X-ray examination.

Regional analgesia is often used in horses and cattle. Operations about the head and limbs may be performed under nerve blocks, sometimes supplemented with chloral hydrate narcosis. In cattle, laparotomies are frequently performed under a paravertebral nerve block, usually with the animal in the standing position. Epidural analgesia also, is sometimes employed.

Since 1951, the pattern of human anæsthetic techniques has been followed, introducing 'balanced anæsthesia'. The dog and cat now receive pre-anæsthetic medication followed by induction and maintenance of anæsthesia in a similar way to the recognised procedures in man. Use is made of ancillary drugs to produce analgesia and relaxation, and respirations are often controlled.

Standard human anæsthetic apparatus is, in general, suitable for cats, dogs, sheep and pigs. In order, however, to extend 'balanced anæsthesia' to cattle and horses, special consideration has had to be given to the 'size of the patient'. Accordingly, special apparatus has been made, which includes a large circle absorber.

Halothane, apart from its cost, is at present the most satisfactory agent for maintaining anæsthesia in large animals and it has the great advantage of being non-explosive.

Anæsthetising different species of animal provides an opportunity to study differences in anatomy, physiology and pharmacological response to drugs. Increasing knowledge should make possible the development of ideal apparatus for all species and all sizes of animal, including apparatus for the mechanical control of respirations.

Induction of anæsthesia in children

Dr. R. J. HAMER HODGES, PORTSMOUTH (Read *in absentia*)

In this country approximately three quarters of a million children under 10 years of age undergo so-called 'minor' anæsthesia annually. The great majority of these children are ear, nose and throat patients, dental or casualty out-patients, or emergency cases. In such patients rapid recovery and immediate return of reflexes is imperative. For these reasons full, adequate, sedative premedication is very often neither safe nor possible.

The anæsthetic management of such children frequently leaves much to be desired. The allegations that anæsthesia is sometimes responsible for post-operative psychic trauma may often be justified. The impact of anæsthesia may be totally unexpected and little understood by the child. Furthermore, lack of sedative premedication is frequently aggravated by the complete absence of psychological preparation. A traditional custom in this country and an almost universal practice in North America is to employ open induction techniques for such casual and minor anæsthetics. These techniques have largely been abandoned in adult practice.

This film demonstrates simply, that psychic trauma need not be an integral part of surgery and anæsthesia, even in unpremedicated children, provided that a sensible and sympathetic approach is made to the child. I believe that intravenous induction techniques are also less frightening than open 'mask' methods. Furthermore, the film demonstrates that intravenous thiopentone can be safe and practical as a method of anæsthesia in so-called 'minor' surgery. The wider application of intravenous techniques together with a sympathetic and sensible approach to pædiatric patients would go far to lessen the psychotraumatic aspects of the operating theatre.

Some pain threshold studies with particular reference to thiopentone

J. CLUTTON-BROCK, BRISTOL

The analgesic drugs commonly used for premedication cause an easily detectable rise in the pain threshold as measured by the spring balance technique[1]. This rise in the pain threshold is promptly

abolished by a small dose of thiopentone given intravenously. The dose of thiopentone required for this antianalgesic effect in healthy adults lies somewhere between 25 and 100mg, but has not been produced by a dose smaller than 15mg. If the dose of thiopentone is increased, the pain threshold rises again, but only after the subject has become unconscious.

Thiopentone also antagonises the analgesia produced by nitrous oxide and oxygen, and causes in certain doses, an increase in the response to a painful stimulus in a patient anæsthetised with nitrous oxide and oxygen. The antianalgesic effect is also produced by pentobarbitone and phenobarbitone; thus, presumably, by all the barbiturates.

The significance of these findings lies, not in the initial effect of any dose of thiopentone – this is transitory – but in the effect of any dose of thiopentone after it has become redistributed. It has been shown[2] that the plasma level of thiopentone, two hours after an intravenous administration, is about a tenth of the initial concentration. If the initial dose was somewhere between 250 and 500mg, then after two hours the plasma concentration will be the same as if 25 to 50mg had just been administered, and this is the sort of dose that produces a profound antianalgesic effect. The concentration of thiopentone in the plasma, after this period, only falls extremely slowly – thus the antianalgesic effect of the initial dose of thiopentone may be very prolonged. Any barbiturate, given for premedication, will also have an antianalgesic effect after the operation. The antanalgesic effect of the barbiturates is not shared by hydroxidione, which may well account for the increase in comfort that has been reported after this drug. A possible neurophysiological explanation of these findings was discussed.

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[2]BRODIE, F. F., MARK, L. C., PAPPER, E. M., LIEF, P. A., BERNSTEIN, E. and ROVENSTINE, E. A. (1950). *J. Pharmacol*, *98*, 85.

The experimental and clinical use of deep hypothermia

D. BENAZON, LONDON

The method devised at Westminster Hospital may be used to produce deep hypothermia to 14°C pharyngeal temperature. At this temperature children and adults may safely withstand at least fifty-two minutes complete circulatory arrest.

The method utilises an extracorporeal pumping circuit and a heat exchanger, but continues to use the lungs as an oxygenator.

During cooling and rewarming 5 per cent carbon dioxide in 95 per cent oxygen is administered. Ventilation is discontinued during the period of arrest.

Temporary changes in pH, total carbon dioxide and serum potassium have been noted, but values return to normal on rewarming.

Independent psychiatric assessment has shown no evidence of cerebral disturbances thus confirming the impression of all those who have been in contact with these patients.

The ventricles may fibrillate, but this is of little importance and defibrillation is easily accomplished even when it has continued for as long as two and a half hours.

Out of fifteen children under seven years old, four never fibrillated, three needed electrical defibrillation and eight reverted spontaneously to normal rhythm.

Using this method a number of different cardiac lesions have been repaired successfully by open cardiac surgery including auricular and ventricular septal defects, mitral and aortic valvular disease, Fallot's tetralogy and ostium primum defects.

It is felt that this is a safe method for use in cardiovascular surgery and that the prospects for extending the period of complete circulatory arrest available are good and further experimental work is continuing on this aspect.

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The full stomach in anæsthesia for trauma

L. WOLFSON, BIRMINGHAM

Although injury delays stomach emptying, postponement of operation necessary on the day of the accident to at least six hours after the previous meal enables the majority of patients to be anæsthetized without gastric reflux during induction.

Special care must be taken to avoid respiratory obstruction in such cases during induction, because it may lead to regurgitation of vomitus and a cuffed endotracheal tube should be inserted in all except brief operations. A head-up tilt of 20° prior to intubation is a promising prophylactic measure, when not contra-indicated.

Special methods of prophylaxis such as passage of a stomach tube (with or without an œsophageal or gastric cuff) prior to induction and endotracheal intubation under local analgesia, have been found unsuitable for routine use in all emergency operations for trauma six or more hours after a meal, as the discomfort to the patient is not justified by the degree of protection afforded.

A stomach tube should be passed before anæsthesia: when operation is too urgent to permit delay; in those few oligæmic patients who cannot be adequately resuscitated before operation; in poor risk patients with coincident disease: because of special dangers of aspiration of vomitus in these groups of cases.

A stomach tube should be passed after induction of anæsthesia if it is not already in place.

At the end of the operation, before deflating the endotracheal cuff: if the injuries permit, the patient should be turned on his side; anæsthesia should be lightened at least to the point of return of reflexes; the stomach should be emptied.

If aspiration of vomitus occurs before the insertion of a cuffed endotracheal tube, oxygenation and tracheo-bronchial toilet must be prompt and vigorous. Equipment for this treatment must be instantly available before anæsthetizing patients at risk, including those with minor injuries.

NEW INVENTIONS

Unless expressly stated by the authors no claim to originality or priority is made and items given under this head include modifications to existing apparatus. The Editor regrets that space considerations make it impossible to publish correspondence on this subject

Oral endotracheal connection

G. H. CAIGER

D. A. S. SICHEL

This small attachment* has been devised by the authors and is clipped to the tongue plate of the Boyle-Davis gag and permits the use of an oral endotracheal tube (*e.g.* for tonsillectomy) with no interference with the surgeon's vision or the operative technique as the tube is held close to the tongue plate. Thus the many disadvantages of a nasal endotracheal tube for this operation are avoided.

This clip is so constructed that it can be slipped on to either side of the tongue plate at the point most suitable to the surgeon. Attached to the part of the clip which holds the appliance to the gag is a hook-like structure beneath which the endotracheal tube passes. A ligature is tied round the vertical limb of the hook before the gag is placed in position in the mouth. The tube is then pressed into position under the hook and the ligature tied. This keeps the surgeon's field of vision clear, and prevents the anæsthetic connections from dragging the tube out. There are no dangerous sharp points to damage tissues in the clip, and it has proved satisfactory in use.

The clip is made in three sizes: the smallest size for children from two to six years, the intermediate size from six to twelve years of age and the large size for young adults is especially useful where a small nasal procedure is combined with tonsillectomy.

*We understand that this attachment may be obtained from Messrs C. F. Thackray Ltd, Welbeck St, W.1. (Editor, *Anæsthesia*.)

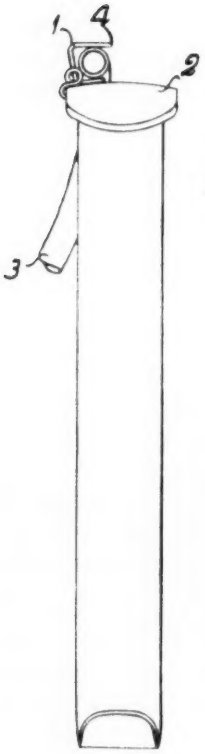


FIG. 1

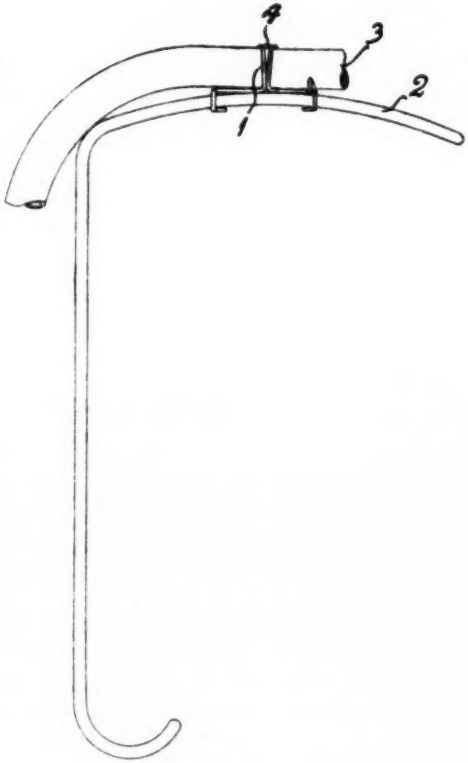


FIG. 2

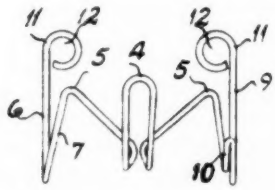


FIG. 3

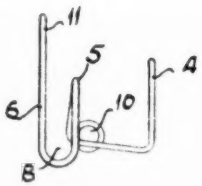


FIG. 4

In some models the ring (10) has been omitted as its function was to attach three clips together by a light chain

A two-way union for double lumen tubes

G. M. J. WHITE

Double lumen tubes were originally designed for bronchospirometric studies but in recent years they have become increasingly popular in anæsthesia for thoracic surgery. They afford independent control of each lung so that the hazard of transbronchial spill-over can be eliminated, and ventilation difficulties in such cases as valvular pneumothorax, bronchial fistula and tension cyst can be avoided. Numerous other advantages with regard to surgical technique are provided.

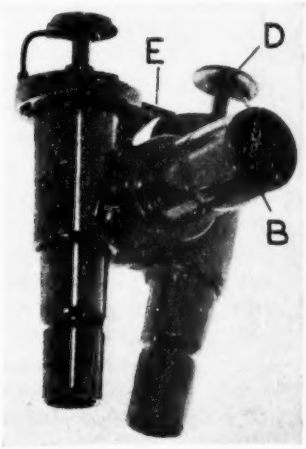


FIG. 1

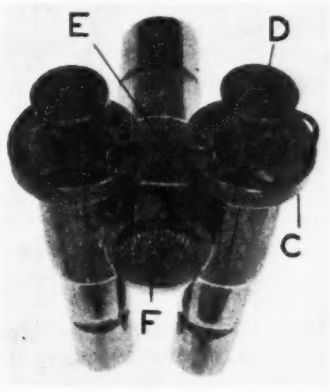
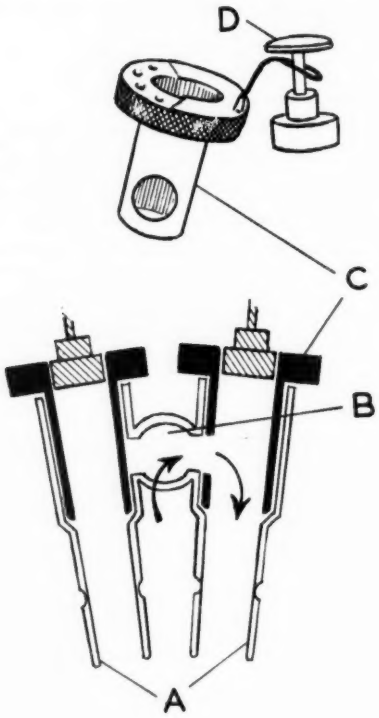


FIG. 2

The two-way union connects the double lumen tube with the catheter connection; it was designed to provide quick and easy control of differential pulmonary ventilation, and satisfies the following requirements: both lungs can be ventilated simultaneously; one lung can be ventilated while the other remains in the inflated or collapsed state; secretions from one lung can be removed while the other lung is being ventilated and fourthly, partial throttling of ventilation of the exposed lung.

The instrument consists of two slightly convergent tubular connections A, fed by a common delivery tube B, placed at right angles. Each connection has an internal rotating cylinder C, which can open, close or partly close the port from the common delivery tube B.



These internal rotating cylinders are fitted with metal stoppers D, which when removed, enable suction catheters to be passed. Removal of the stoppers also allows lung gases to be expelled, thus permitting collapse of the exposed lung. A common spring E, which is fixed by a knurled finger screw F, operates on the flange of the internal rotating cylinders and retains them within the connection. The spring engages with dimples on the flanges so that any particular rotatory position of the cylinders is maintained. A clockwise rotation closes the connection and vice versa while the two intermediate positions provide partial throttling.

An exposed lung is collapsed by closing the corresponding connection and removing the stopper to allow exit of the lung gases. This is particularly useful in such cases as pneumonectomy in that it provides more operating room within the thorax. However, during lobectomy or segmental resections, partial inflation of the exposed lung is beneficial in that it reduces lung volume and lung movement but at the same time delineates pulmonary lobes and segments and

allows a certain amount of gaseous interchange to take place. Such conditions are provided by the throttling mechanism.

Gas flow resistances were measured for the two-way union alone and then connected to a Carlens catheter (FG 39). The results are seen in the table and may be compared with flow resistances for a size 8 Magill endotracheal tube of length 31cm and a Magill connector size 6A the smallest diameter of which is the same as that of either connector of the two-way union.[1]

LITRES/MIN	5	10	15	20	25
Two-way union					
Both connectors open	0.4	1.5	3.3	5.7	9
One connector open	0.9	3.5	8.3	15	24
Two-way union plus Carlens catheter	3.2	10.3	23	38	–
Size 8 magill endotracheal tube	–	5	7	10	15
Magill connector size 6A	2	3	4	5	7

Gas flow resistances in MM *water*

The instrument can be readily dismantled for cleaning by undoing the knurled finger screw F, removing the spring E and withdrawing the internal rotating cylinder C which are interchangeable. It was designed by Mr R. Salt of the Nuffield Department of Anaesthetics, Oxford and the writer and is made in stainless steel in one size only and this fits the three sizes of double lumen tubes FG 37, 39, 41. It is manufactured by and obtainable from H. G. East & Co, Oxford.

Acknowledgement

I am indebted to Miss M. C. McLarty for the drawing and to Dr H. G. Epstein for carrying out flow rate measurements.

Reference

[1] *Physics for the Anæsthetist, Chap. XIII.*

Automatic unidirectional flow valve

D. F. REES

The valve consists of a brass cylinder with four exhaust ports in line (two are visible in FIG. 1 A). The two bulges represent bypass passages with a port at each end (B, B_1 and C, C_1), which carry the inspired gases past the piston.

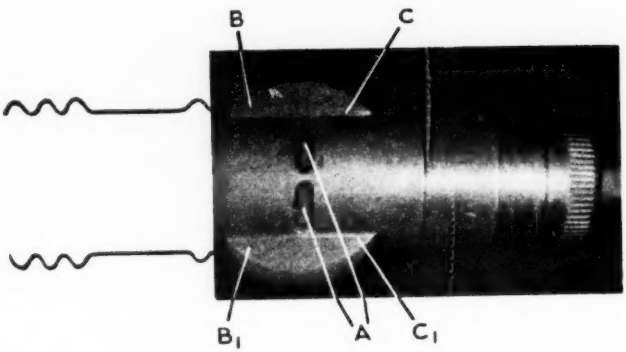


FIG. 1.

The freely floating piston is turned from compressed fibre, and in FIG. 2 showing the inspiratory position, is seen occluding the exhaust ports.

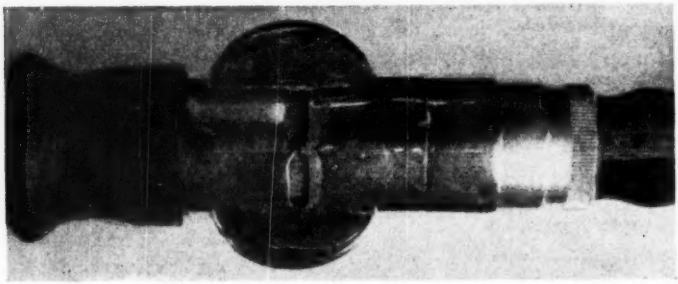


FIG. 2 Inspiratory position

The valve is dimensioned to take standard BOC masks, angle and tube connecting pieces.

During controlled or assisted respiration, the clearance of the piston is such that there is sufficient blow back via the exhaust ports to prevent harmful pressures being built up in the system.

With spontaneous respiration, this will also occur provided that the valve is horizontal or sloping up to the patient, and the gas flow is adjusted to fill the bag and push the piston over the exhaust ports at the end of expiration. In practice this has meant from 5 – 9 litres per min total flow, depending on the volume of excursion.

It may be noticed that the Dewsbury valve exactly duplicates the functions of the Rubin valve. When the latter was published, the first prototype Dewsbury valve had been completed and shown to be

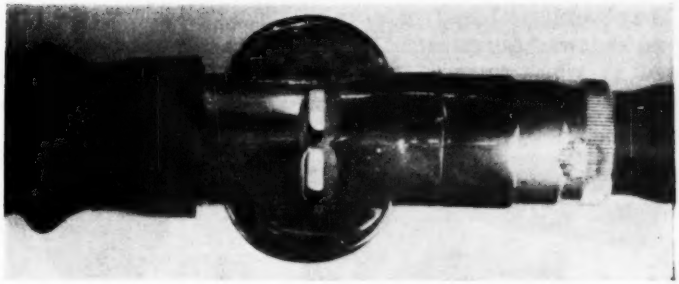


FIG. 3 Expiratory position

a practical approach to the problems of the automatic non-rebreathing valve.

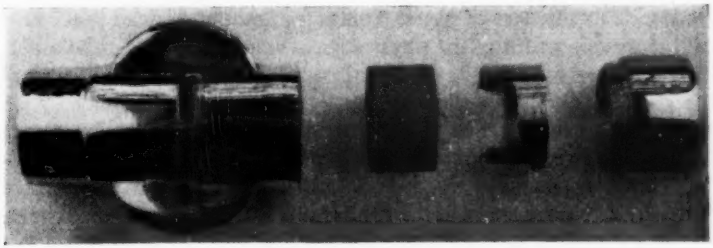


FIG. 4

Exploded view showing cylinder | Piston | Distal piston stop | End piece

The photographs are of the fourth prototype, the fruits of two years development.

My thanks are due to Dr A. D. Charnley for the photographs, and to Charles F. Thackray Ltd. of Leeds, who constructed the valve.

Perspex endotracheal adapter

H. H. SAMSON

Reduction of dead space and resistance to the flow of gases in general anæsthesia is desirable especially in patients undergoing protracted operations and in those who may not be able to compensate adequately, *viz*, young children, the aged and the seriously ill.

The physiological dead space can be reduced considerably by means of an endotracheal catheter. However, currently used attachments (FIG. 1) which connect the endotracheal adapter to the anæsthetic machine, create unphysiological dead space which is redundant

FIG. 1

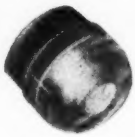


FIG. 2

even when the rubber tubing is considerably shortened. Such devices still contain 6 ml or more of unnecessary dead space and also offer resistance to the flow of gases. This extra amount of dead space may well represent a fifth of the physiological dead space in a child and is distributed as follows: in the metal connection; within the lumen of the rubber tubing; in the lumen of the adapter, especially in the expanded portion and in the metal tube proximal to the expiratory valve (E) shown in cross-section in the diagram. (FIG 5).

Metal adapters, into which endotracheal connections are inserted (this obviates dead space in the rubber tubing), are not new. However, the adapter illustrated by FIG. 2 is made of perspex and eliminates the dead space described above. This is effected by inserting the

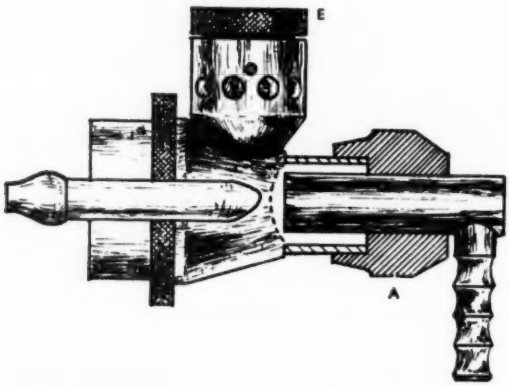


FIG. 5 Cross-section of adapter and metal tube proximal to valve.

angled connection firmly into the adapter so that the end of the smooth limb protrudes from the adapter (FIGS 3 and 5 (A)). The adapter being made of perspex is light and slightly resilient. This provides a better fit. This adapter has been designed to fit a MIE (FIG. 4) or Boyle's attachment, and a Rowbotham connection which

FIG. 3

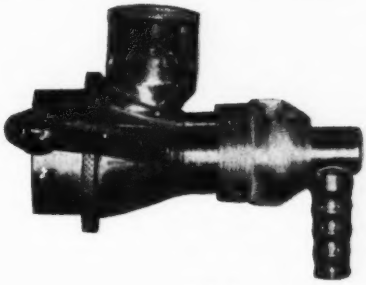


FIG. 4

has a straight limb. Curved endotracheal connections which have been shown to offer less resistance[1] to the flow of gases, can be made to fit the adapter.

The adapter is durable and may be sterilised by boiling or autoclaving. It is not affected by the vapours of trichlorethylene or halothane.

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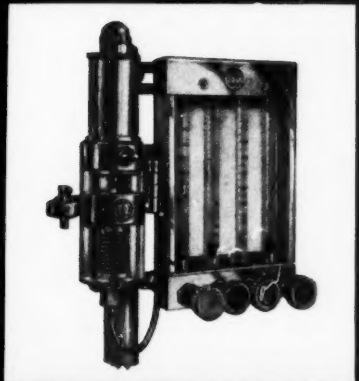

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Correspondence

SPINAL ANALGESIA AND UNRELATED PARAPLEGIA

The Editor, *Anæsthesia*

Sir,

I was interested to read the article on spinal analgesia with unrelated paraplegia (R. A. L. Leatherdale (1959), *Anæsthesia, 14*, 274).

In January of this year I gave spinal analgesia, using heavy lignocaine, between L.3 and 4, to a fit young man of 25 years. He had had a Ewing's sarcoma excised from the lower end of his tibia and this was one of a series of operations during which the gap was being filled with a pedicle graft, but it was his first spinal block. There was no evidence of secondaries.

Next day the patient complained of headache and also back pain in the lumbar region, radiating to the flanks. The symptoms were attributed by all, including the patient, to the spinal analgesia. The headache cleared in two to three days but the back pain remained. There was no weakness of bladder or legs and X-ray revealed no abnormality. It was thought that possibly the rigid way in which he lay in bed may have been aggravating the pain and he did improve slightly with exercise. During the remainder of his operations, which were performed under general anæsthesia, the pain fluctuated but never entirely disappeared.

He was discharged but returned in three months with more severe pain and this time the X-ray showed collapse of the body of L.1, the appearances being more typical of TB than secondaries but, as his general condition was deteriorating, assumed to be the latter. His relations then took him away and we have lost trace. It is difficult to follow up patients adequately here and as I am keeping records of all spinal and epidural complications this is an example of how the record would have been weighted against spinals if the patient had not returned.

Yours faithfully,
F. N. Prior
Christian Medical College
Ludhiana, Punjab, India

BRONCHOSPASM

The Editor, *Anæsthesia*

Sir,

The interesting article in the October issue by Drs Young and Mendel, prompts me to describe a technique of pre-medication which I have used for three years and which was proved of value in avoiding bronchospasm during anæsthesia.

I have used promezathine hydrochloride as a nocturnal sedative in acute and chronic asthmatics encountered in general practice and found it to be effective more often than any of the more traditional drugs, so that it seemed reasonable to try it for the same sort of patient presenting for operation.

I now use it routinely for the following groups of patients: all patients with a history of 'wheezy attacks' or frank asthma; all patients with bronchospasm or

with marked prolongation of expiratory sounds on auscultation; chronic bronchitics; emphysematous patients; patients with upper respiratory infection; heavy smokers, *i.e.* smoking more than ten cigarettes daily.

Where the chest condition warrants it and where it is possible to postpone surgery, I order promezathine hydrochloride 50mg nightly by mouth for one week and also prescribe vigorous breathing exercises and postural drainage where needed. Two hours before operation, the patient is encouraged to do breathing exercises and to expectorate his sputum. He is then given an intramuscular injection of 50mg promezathine hydrochloride with 50mg pethilorfan followed by atropine 1/100gr forty minutes later. The old, frail and the ill may have the pethilorfan reduced or omitted.

For patients without sputum and only mild degrees of bronchospasm and for the heavy smokers, it is sufficient usually to give one dose of 50 to 10mg promezathine hydrochloride by mouth twenty four hours pre-operatively. On the day of operation I include a further 50mg intramuscularly as part of the pre-medication.

Since using this technique I have never had an induction laryngo- or bronchospasm due to thiopentone, whereas previously I have noted these alarming complications fairly frequently. Whilst I cannot quote statistics, it is my impression that it is necessary to use aminophylline to relieve bronchospasm during operation far less often than I used to. In fact I have used aminophylline only three times in three years.

The patients arrive in theatre drowsy, though capable of being roused, and when visited post-operatively report that they remember little or nothing of the day of operation and have neither vomited nor coughed. The chronic bronchitic or emphysematous patients frequently volunteer the information that 'my chest never felt better, Doctor'.

Yours faithfully

Margaret C. Watkinson

COLLAPSE AFTER HALOTHANE

The Editor, *Anæsthesia*

Sir,

The reports of cases that have collapsed after halothane anæsthesia whilst using the fully closed circuit, have been due in my opinion to two simple reasons.

In the cases of Young and Lodge[1], the nitrous oxide was introduced into the circuit without turning off the soda lime and the oxygen was not increased. The soda lime must be turned off with the halothane[2]. Therefore, the resulting hypoxia gave rise to serious cardiac embarrassment. As soon as the gas flow was increased to 5 litres nitrous oxide plus 2 litres oxygen with the soda lime off all was well. This weaning of the cases on the semi-open circuit is still the method recommended after three years daily use with not one case like these reported.

The case reported by ApIvor[3], in my opinion was probably too deep too long, as persistent cardiac irregularities and a marked fall in blood pressure are classical symptoms. On these occasions it is suggested that the halothane be turned off and the patient inflated with pure oxygen. A return to closed halothane and oxygen may then be made. Should, however, the irregularities return, then a change to ether is recommended. In my experience this manœuvre is very rarely done, although an essential safety factor. During long operations the concentration of any inhalation anæsthetic should always be reduced as time goes on, because

surgical shock lowers the patient's resistance and the body becomes saturated. This reduction in concentration applies particularly to the ill toxic cases and elderly patients.

There has been one non-fatal serious cardiac embarrassment due, I am sure, to hypoxia. It was an old man, who had undergone a lengthy operation for the removal of his prostate, and had been given halothane plus oxygen in the closed circuit without incident up till the last skin suture. The mask was then removed without any previous weaning on the semi-open circuit. His respiratory excursion, which was previously adequate on the high oxygen content in the closed circuit was not sufficient for the sudden drop in oxygen percentage obtainable from the atmosphere. On the other hand for very short procedures the halothane may be given up to the end, and the respirations will quickly adjust themselves.

These reports have been most valuable but I am still convinced that to wean cases through 5 litres nitrous oxide plus 3 litres oxygen on the semi-open circuit not only safeguards them from 'cyclopropane shock,' but enables the halothane to be turned off well before the end of the operation.

Yours faithfully,
H. R. Marrett

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ANÆSTHESIA FOR FREDET-RAMMSTEDT'S OPERATION

The Editor, *Anæsthesia*

Sir,

We have read with considerable interest the article on the above subject by Drs Booth, Nisbet and Wilson in the October 1959 issue of *Anæsthesia*.

We are grateful that the authors end their paper on a note of caution regarding the use of thiopentone/relaxant induction in these cases. Nevertheless, we feel that the advice they give to intubate the conscious infant of under seven pounds may be applied in many instances in the more robust child over this weight. We believe that an attempt should be made to perform this in every case and a surprising number of quite strong infants can be intubated in this manner atraumatically. Further, we consider that it is a hazardous procedure to abolish respiration in these infants prior to intubation for the following reasons.

Firstly, even in expert hands there may be difficulty in intubating the trachea and if delay is experienced it is well known that inflation of the lungs via a face mask may be ineffective and grave hypoxia ensue.

Secondly, there remains the ever present danger of regurgitation of gastric contents, however meticulous the pre-operative preparation may have been. Therefore, we would advocate the induction of anæsthesia by inhalational methods in those cases where it is found impracticable to intubate the trachea with the infant conscious.

Currently one of us (S.H.S.L.) is investigating the following anæsthetic technique as an alternative to that previously described by us[1].

With the patient conscious, gentle insertion of the laryngoscope is carried out with aspiration of secretions (if any) from the mouth and oro-pharynx. Tracheal intubation is performed if it can be done easily and atraumatically. Anæsthesia is

then accomplished by nitrous oxide–oxygen using a modified Ayre's T piece and relaxation is obtained by intramuscular or intravenous injections of succinylcholine. Respiration is controlled.

Alternatively, if intubation in the conscious patient is not found practicable then the passage of the endo-tracheal tube is facilitated by halothane added to a nitrous oxide – oxygen mixture. Following intubation the halothane is discontinued and anæsthesia is maintained as before.

In the small number of cases studied this has been a satisfactory alternative technique of anæsthesia for the Fredet-Rammstedt's operation.

Yours faithfully,
S. H. S. Love
G. W. Black

[1]BLACK, G. W. and LOVE, S. H. S. (1957). *Anæsthesia*, **12**, 430.

Obituary

Dr George F. Rawdon Smith, TD, MD, FFARCS, died at his home in Little Gaddesden, Hertfordshire in October last at the age of 78. He had returned to the south of England some years ago after being for many years a general practitioner of distinction in Liverpool and an Honorary Anæsthetist to the Liverpool Royal Infirmary, the Royal Southern Hospital, Hospital for Women and Dental Hospital. He was for many years Lecturer in Dental Anæsthetics at the University of Liverpool and was the author of a text book 'Dental Anæsthesia' in the Outlines of Dental Science series published in 1926. He obtained his DA in 1935 and was elected a Fellow of the Faculty of Anæsthetists in 1948. He had the distinction of being elected President of the Liverpool Medical Institution in 1946.

George Smith was one of the last of the old generation of general practitioner-anæsthetists who, in an honorary capacity, staffed our General and Teaching Hospitals from the first introduction of anæsthetics until the inauguration of the Health Service in 1948. They took the strain of surgical lists and trained, as students, the men and women who were to progress further with modern techniques and inaugurate the anæsthesia of today. He was always interested in the teaching of students and wrote an article on 'The teaching of anæsthesia'. He was a cultered man of wide interests with a distinguished army and Territorial Army career – holding the rank of Colone lin the 88th Division T.A. He had a keen scholarly mind and kept abreast of the developments in anæsthesia of his day. He is long remembered by his colleagues for his endotracheal techniques with the Kelly machine and his use of the Oxford Vaporiser as well as for his series of investigations at the Women's Hospital in the very early days of intravenous anæsthesia and in the premedication of children. He was a Senior Fellow of the Association of Anæsthetists.

Having worked with him for many years, it gives me great pleasure to pay tribute to him as teacher, colleague and friend.

FANNIE S. BALL

Book Reviews

AUTOMATIC VENTILATION OF THE LUNGS

W. W. MUSHIN, L. RENDELL-BAKER and P. W. THOMPSON

Pp. xvi+349. Figs. 191. Blackwell Scientific Publications, Oxford. Price 47s. 6d.

Controlled respiration was originally introduced in 1934 to provide a tranquil operating field in abdominal surgery. Its use during anæsthesia was soon extended to thoracic surgery while the introduction of the specific muscle relaxants widened the scope of the technique so greatly that it is now a commonplace for many cases on an ordinary hospital operating list. Recently, controlled respiration has invaded the field of medicine and has proved life-saving in certain cases of bulbar poliomyelitis, tetanus, gas poisoning, *etc.*

The original method of providing intermittent positive pressure by manual compression of the reservoir bag of an anæsthetic apparatus proved extremely effective and is still in constant use. Automation has, however, proceeded apace and a very large number of machines are now on the market to provide automatic ventilation; many being able to incorporate an adjustable negative pressure phase in the respiratory cycle. The difficulty facing the practical anæsthetist has been to choose the most suitable apparatus and his choice has necessarily been influenced by the advertising and sales-talk of the various manufacturers. This unsatisfactory position should be altered by the timely publication of *Automatic Ventilation* as for the first time the whole subject has been dealt with in a scientific manner. The elements of respiratory physiology are clearly set out and from this basis the principles of controlled breathing are elucidated. Practically every available ventilator adaptable to anæsthesia is described and illustrated under classified headings so that accurate comparison is possible. The only omissions are those respirators which act by applying intermittent pressure to the body surface—either positive as in the cuirass type, or negative as in 'tank' respirators. It is true that these have little direct interest for the anæsthetist, but he may well be called in to advise on their correct functioning and adjustment. Their inclusion would have made this admirable monograph complete.

ANESTHESIA FOR INFANTS AND CHILDREN

ROBERT M. SMITH

Pp. 481. Figs 182. The C. V. Mosby Co., St. Louis, U.S.A.

Distributed in Great Britain by Henry Kimpton, London. Price 90s.

This is a new book on pædiatric anæsthesia by the head of the anæsthesia service of the Children's Medical Centre, Boston, Mass., who has had a wide experience in this field.

The text is written from a practical aspect, the many theoretical considerations being discussed adequately, but without undue emphasis. The line taken throughout is thoroughly sound and the various techniques are described with sufficient detail to enable a junior anæsthetist to follow them without difficulty. It is refreshing to find that simple methods, such as open ether, are adequately dealt with and not condemned as obsolete. Divinyl ether is recommended for induction

instead of the more usual ethyl chloride used in this country. It is interesting to note that halothane, 'should only be used in a semi-closed technique with a vaporizer calibrated between 0 and 2 per cent'. The chapter on pre-operative medication is useful, but it seems a pity that trimeprazine is not mentioned. Anæsthesia for thoracic surgery in children is well described and includes hypothermic and bypass techniques in open heart operations. There is an excellent section on Respiratory Physiology in Infants and Children by Dr Charles D. Cook, Assistant Professor of Pædiatrics in the Harvard Medical School. The difficult subjects of fluid therapy and blood replacement are discussed in a most useful chapter. There are a few errors such as that one of the disadvantages of trichlorethylene is that it combines with carbon dioxide absorbents to form phosphoric acid. The toxic compound is actually dichloracetylene.

Most of the illustrations are exceptionally clear reproductions of photographs and the book is beautifully printed and bound.

RESUSCITATION OF THE UNCONSCIOUS VICTIM

PETER SAFAR and MARTIN C. MCMAHON

Pp. 79. Charles C. Thomas, Springfield U.S.A. and in the British Commonwealth, Blackwell Scientific Publications Ltd. Oxford. Price 12s. 6d.

This useful manual intended for lay rescue workers has been written by the Chief of the Department of Anesthesiology, Baltimore City Hospital, and by the Captain of the Baltimore Fire Department Ambulance Service. It deals with simple methods of ventilating unconscious patients who have stopped breathing or whose airways are obstructed. Emphasis is placed on the superiority of intermittent positive pressure respiration (by the rescuer's expirations, atmospheric air or oxygen) over any type of manual artificial respiration. The instructions given are clear, easily understood and should prove of great service. The only slight criticism is that the artist in his line drawings has endeavoured to show the importance of extending the head but has rather overreached himself so that the extreme hyperextension shown in some cases might defeat its object by actually producing respiratory obstruction. The book is well printed and bound in a stout paper cover.

PRINCIPLES AND PRACTICE OF OBSTETRIC ANÆSTHESIA

J. SELWYN CRAWFORD

Pp. 128. Blackwell Scientific Publications. Oxford. Price 20s.

In the course of an admirable introduction Dr Crawford remarks that there is no modern British textbook of obstetric anæsthesia. He has made a very successful effort to remedy this deficiency and discusses the subject under the following chapter-headings:—The Mother—The Placenta—Analgesia—Forceps Delivery and Caesarian Section—Other Indications for Anæsthesia—The Child—Equipment and Administration—Epilogue. The author considers all these factors with sane commonsense and is clearly informed of the latest relevant research. He has had the advantage of working on both sides of the Atlantic and, as is well known, conditions in North America differ radically from those obtaining in Great Britain—for example, the midwife system is virtually non-existent and

active participation by the mother in her labour is not generally observed. The balanced viewpoint which Dr Crawford achieved by this wide experience is matched by real literary style which makes the book a pleasure to read. The comparatively short section on infant resuscitation is first class and is as valuable in listing proved methods as in debunking useless and even dangerous survivals of the Sairey Gamp era. It is rather a pity that actual techniques such as pudendal block are not described in sufficient detail to be used as a guide. A considerable number of modern references is given at the end of each chapter and there is a short and not very comprehensive index. Obstetricians and paediatricians as well as anæsthetists will find much that is informative and stimulating in this book.

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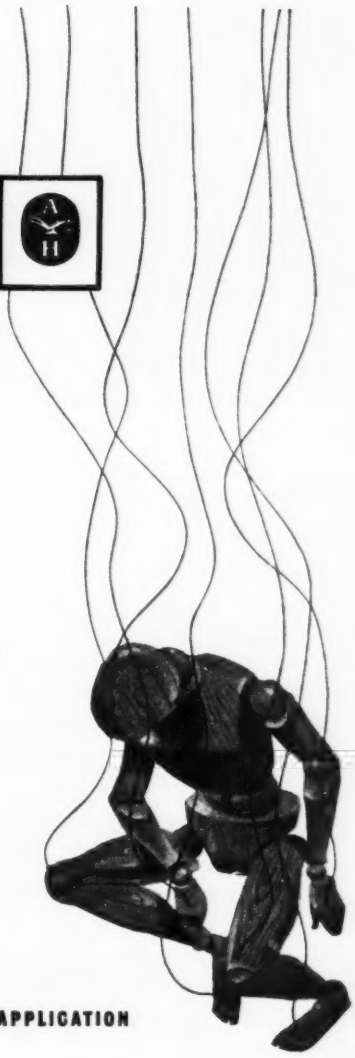

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Association News

The Annual Meeting of the Association was held in Stratford upon Avon on 22 – 23 October. Amongst the three hundred Fellows and Members present were anæsthetists from Denmark, Iran, Malaya, the Netherlands, Portugal, South Africa, Sweden and the United States of America.

The annual business meeting was held on Thursday afternoon. The President, Professor T. Cecil Gray, presented the Report of Council for 1958 – 9 which will be circulated later. The following Officers and Members of Council were elected:

PRESIDENT	Dr Ronald Jarman, *London*
VICE-PRESIDENT	Dr R. P. W. Shackleton, *Southampton*
HONORARY TREASURER	Dr Vernon F. Hall, *London*
HONORARY SECRETARY	Dr John Beard, *London*
EDITOR	Dr C. Langton Hewer, *London*
ASSISTANT EDITOR	Dr R. Blair Gould, *London*
MEMBERS OF COUNCIL	Dr Russell M. Davies, *East Grinstead*
	Dr J. W. Dundee, *Belfast*
	Dr J. Alfred Lee, *Southend-on-Sea*
	Dr H. J. V. Morton, *London*
	Dr J. B. Wyman, *London*

Professor T. Cecil Gray, as Immediate Past President, becomes a Vice-President.

Dr John Gillies, Sir Robert Macintosh and Mr A. D. Marston were elected Honorary Fellows.

Dr M. de Faria, Brazil: Dr J. L. C. Fernando, Ceylon: Dr W. Hugin, Switzerland: Dr O. Ribeiro, Brazil: Dr V. Smolnikoff, U.S.S.R. and Dr E. Lopes Soares, Portugal were elected Corresponding Fellows.

An addition to the Rules to allow for the election of Associate Fellows was approved. Such Associate Fellows may now be elected from registered medical practitioners and others who, while interested in anæsthesia, are not engaged in the medical practice of anæsthetics. They will have no voting rights.

The proposal to increase the subscription of Ordinary Fellows, Associate Fellows and Members to £3 . 3 . 0 per annum was also approved, the annual subscription for Associates remaining at £2 . 0 . 0.

The second John Snow Memorial Lecture was delivered by Dr W. Grey Walter on 'The control of consciousness'.

The Annual Dinner was attended by 317 Fellows, Members and guests. The toast of the Association was proposed by the Dowager Marchioness of Reading, GBE in a gracious and witty speech, to which the President, Dr Ronald Jarman, replied. The Immediate Past President, Professor T. Cecil Gray, proposed the health of the guests, and Lieut.-Colonel Sir Fordham Flower, OBE, DL, replied to this toast.

Papers were read at the morning and afternoon sessions on Friday: summaries of these will be found elsewhere in this issue. A trade exhibition was held throughout the Meeting, which concluded with an appropriate visit to the Shakespeare Memorial Theatre – 'All's Well that Ends Well '.

Essay Prize. The Council of the Association of Anæsthetists of Great Britain and Ireland is at present especially interested in matters which may improve the efficacy and safety of dental anæsthesia.

CANADIAN ANÆSTHETISTS' SOCIETY JOURNAL

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It has, therefore, been resolved to offer a prize of two hundred guineas for the best essay based upon new and original work related to the subject of general anæsthesia for dental surgery. The Council would especially welcome essays dealing with the morbidity and the safe practice of general anæsthesia in the dental chair.

Essays should be submitted to the Honorary Secretary, Association of Anæsthetists of Great Britain and Ireland, 47 Lincoln's Inn Fields, London WC2, on or before 1 March 1961. Intending candidates should apply to the Honorary Secretary at the above address for the regulations governing the submission of essays.

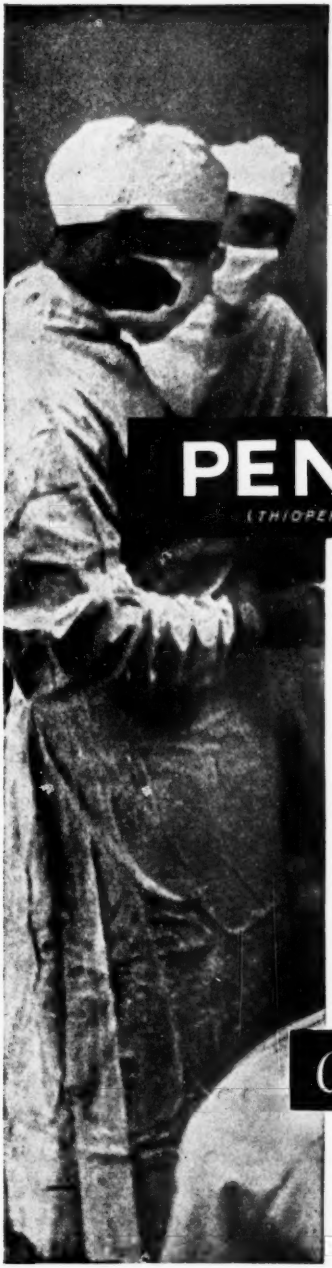
The **Anæsthetists of the North-east Metropolitan Region** held their autumn meeting on Saturday 1 November 1959. A most interesting tour of the works of the de Havilland Aircraft Co. at Hatfield was arranged in the morning. After lunch at Hill End Hospital, St Albans, Dr R. I. Ballantine gave a talk on anæsthetic problems involved in the separation of conjoined twins. This was illustrated with some excellent slides. The last hour was occupied by the efforts of a small panel to answer questions proposed by the audience.

The **Liverpool Society of Anæsthetists** has arranged the following programme for 1960.

FRIDAY 22 JANUARY 8PM	Ordinary general meeting at Liverpool Medical Institution Dr J. Alfred Lee: Experiences of anæsthesia in a provincial hospital.
WEDNESDAY 24 FEBRUARY 8PM	Ordinary general meeting at Liverpool Medical Institution Papers to be presented by registrars
THURSDAY 17 MARCH 8PM	Joint meeting with Liverpool Medical Institution at Liverpool Medical Institution Hypothermia in Cardiac Surgery Professor T. C. Gray. Dr Charles McKendrick. Mr L. J. Temple. Dr J. C. Richardson. Dr I. C. Geddes.
THURSDAY 28 APRIL	Combined meeting with Manchester Medical Society in Manchester Dr. G. N. Mulliner: Anæsthesia and Body Temperature. Dr J. C. Richardson: New Developments in Anæsthesia for Cardiac Surgery.
WEDNESDAY 18 MAY 8PM	Twenty-eighth annual general meeting at Liverpool Medical Institution.

The **Section of Anæsthetics of the Manchester Medical Society** has arranged the following programme for 1960:

THURSDAY 28 JANUARY	Registrars' meeting Dr I. Hallack. Dr B. J. Muir.



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THURSDAY 25 FEBRUARY	Symposium on Dental Anæsthesia Dr T. Dinsdale. Dr W. Ken Jones. Mr G. Ashcroft.
THURSDAY 31 MARCH	Dr Frankis T. Evans: Mistakes I have made.
THURSDAY 28 APRIL	Joint meeting with the Liverpool Society of Anæsthetists at Manchester Dr G. N. Mulliner: Anæsthesia and Body Temperature. Dr J. C. Richardson: New Developments in Anæsthesia for Cardiac Surgery.

The **Anæsthetic Section of the Royal Society of Medicine** is able to offer a prize of £30 for the best paper written by a medical practitioner of Senior Registrar or Registrar status in the National Health Service holding a clinical appointment in anæsthesia. Fellowship of the Royal Society of Medicine is not necessary for entry. The subject will be of the author's choice but must be connected with anæsthesia. All papers for the 1961 award must be submitted by 1 January 1961. Further details and rules of the prize can be obtained from the Honorary Secretary, Anæsthetic Section, Royal Society of Medicine, 1 Wimpole Street, London W.1

CORRECTION

Vol 14. No 4, October 1959 p. 362 line 5 – 'dose of 2mg/lb (1mg/kg)' should read 'dose of 1mg/lb (2mg/kg)'.

Faculty News

The next **course of lectures, demonstrations and tutorials** will be held from 8 – 19 February 1960.

The **Annual Meeting** of the Faculty will be held on 16 March 1960 and on the same day the **Joseph Clover Lecture** will be delivered by Dr J Alfred Lee FFARCS. The **Twelfth Anniversary Dinner** will follow in the evening.

At the last **Final Examination for the Fellowship in the Faculty of Anæsthetists,** 140 candidates presented themselves, 35 of which acquitted themselves satisfactorily. Two of these had not, at the time of writing, complied with the regulations. The remaining successful candidates were:

Davis, Fredrick John
Tunstall, Michael Eric
Brookes, Robert Charles
Ordish, Pamela Mary
Parbrook, Geoffrey Donald
Franklin, Clifford Bruton
Toner, John Stannard
Som, Murari Mohon
Briffa, Joseph Edward
Fraser-Jones, John
Levitt, Mendel
Chippindale, Lorna
Egginton, Michael John
Greenan, Joseph
Ahmed, Nazir
Nisbet, Hugh Ian Armstrong
Weerackody, Upali
Casserley, Joan Dorothy Anne
Cooke, Michael
Davidson, Lois Ruth
Fitton, Edward Paul
Ball, David Reginald
Bennett, Edward Dick Luddington
Bryson, Thomas Henry Learmont
Fernando, Lionel Clarence
Frost, Phillida Mary
Morgan, John Gilvier
Wilson, John
Cole, Peter Vernon
Holderness, Margaret Carleton
Knight, Peter Frederick
Fisher, Frank
Stoddart, Joseph Charles

The **Burroughs Wellcome Fund** in the U.S.A. has founded a Fellowship in Anæsthesia at the University of California Medical Center, Los Angeles, to give a British physician in training in anæsthetics the opportunity of the cultural, academic and professional experience of an American university and to increase the understanding of anæsthetics between the physicians of the United States and Great Britain. The value of the Fellowship is 4,000 dollars p.a., including travelling expenses. This amount has been found by the first Fellow to be adequate to cover normal requirements. The Fellowship has been founded for three years to support three Fellows for one year each, and the Fellows are expected to return to Britain upon the completion of the terms of their respective Fellowships. The Fellowship is for both clinical and research work.

Anæsthetists who have passed at least the Primary Examination for the FFARCS are invited to apply for the third annual award. The Fellow appointed will be expected to take up his duties as soon as possible after 1 September 1960. Applications, giving details of qualifications and experience (including research experience, if any) and the names of two persons to whom reference may be made if necessary, should be sent to R. S. Johnson-Gilbert, Esq., MA, Secretary, Faculty of Anæsthetists, Royal College of Surgeons of England, Lincoln's Inn Fields, London WC2 not later than Monday 8 February 1960.

Commonwealth and Foreign News

The **Australian Society of Anæsthetists** has circulated a most interesting newsletter compiled by Dr Brian Crawshaw. It contains a disquieting account of how four separate anæsthetists in different theatres of the Sydney Hospital got into difficulties with cyanotic patients in spite of using 100 per cent 'oxygen'. It

subsequently transpired that a batch of 'oxygen' cylinders had been delivered which, in fact, contained compressed air. At the time of writing no explanation had been forthcoming from the suppliers. It speaks well for the vigilance of the anæsthetists that no fatalities occurred and readers might well remember this possibility when faced with unexpected cyanosis.

The **Society of Anæsthetists of Hong Kong** held a combined meeting with the Chinese Medical Association on 24 September 1959 on the subject of 'Shock'. Dr R. Barnes spoke on 'Shock in Medical Conditions'. Mr J. Fung on 'Shock in Surgical Conditions', and Dr Nancy Butt on 'Anæsthesia in the shocked patient'.

The sixth Congress of **The Scandinavian Society of Anæsthesiologists** will be held in Gothenburg, Sweden 30 June – 2 July 1960.

The main subjects of the Congress will be: Pædiatric anæsthesia. Pre- and post-operative disturbances of circulation. Post-graduate training of anæsthesiologists. The three main subjects will be dealt with both in panel discussions and in papers, but papers on other subjects within the field of anæsthesiology may also be submitted. Entries of papers, accompanied by manuscript or an abstract giving full details of the subject, should be made to the Secretary-General before April 1960. The official Congress languages will be Swedish, Norwegian, Finnish and Danish; but, if desired, papers may be read in English or German. Further particulars as to the scientific and social programmes of the Congress, entries for participations, *etc.*, will be given in due course.

The tenth **French Congress of Anæsthesiology** was held at Lyon from 9 – 12 July 1957 and was attended by Dr Alex C. Forrester as representative of the Association of Anæsthetists. During the Congress, Prof. Voorch was appointed to the Chair of Anæsthesia in Paris. He is the first anæsthetist to hold this office, the previous holders being surgeons. It is gratifying to learn that the status of French anæsthetists is steadily improving although some nurse-anæsthetists still remain. Post-graduate teaching is also progressing, an annual course of one hundred lectures being given, seventy by anæsthetists. The specialist examination somewhat resembles the FFARCS and is held in two parts (1) basic and (2) clinical. Anæsthetists now take a major part in the examination of candidates.

The **Association of European Anæsthetists** held its second Congress in Lyon on 12 July 1959.

Communications were given by: J. Bonica (U.S.A.): 'The public relations of the anæsthesiologist'. G. Organe: 'The organisation of the anæsthetic services in Great Britain.' Dr Martin: 'The instruction programme for the anæsthesiologist in the U.S.A.'

There was a discussion on the place of the anæsthetist in modern medicine.

The **Mexican Society of Anesthesiologists** is holding its eighth congress jointly with the fifth Latin-American congress at the University City in Mexico DF, in November 1960. Fellows and Members of the Association of Anæsthetists of Great Britain & Ireland are invited to attend or to submit scientific articles. All correspondence should be addressed to Al Apartado Postal 13649, Admon, 45, Mexico 1, DF.

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The sixth Congress of **The Scandinavian Society of Anæsthesiologists** will be held in Gothenburg, Sweden 30 June – 2 July 1960.

The main subjects of the Congress will be: Pædiatric anæsthesia. Pre- and post-operative disturbances of circulation. Post-graduate training of anæsthesiologists. The three main subjects will be dealt with both in panel discussions and in papers, but papers on other subjects within the field of anæsthesiology may also be submitted. Entries of papers, accompanied by manuscript or an abstract giving full details of the subject, should be made to the Secretary-General before April 1960. The official Congress languages will be Swedish, Norwegian, Finnish and Danish; but, if desired, papers may be read in English or German. Further particulars as to the scientific and social programmes of the Congress, entries for participations, *etc.*, will be given in due course.

The tenth **French Congress of Anæsthesiology** was held at Lyon from 9 – 12 July 1957 and was attended by Dr Alex C. Forrester as representative of the Association of Anæsthetists. During the Congress, Prof. Voorch was appointed to the Chair of Anæsthesia in Paris. He is the first anæsthetist to hold this office, the previous holders being surgeons. It is gratifying to learn that the status of French anæsthetists is steadily improving although some nurse-anæsthetists still remain. Post-graduate teaching is also progressing, an annual course of one hundred lectures being given, seventy by anæsthetists. The specialist examination somewhat resembles the FFARCS and is held in two parts (1) basic and (2) clinical. Anæsthetists now take a major part in the examination of candidates.

The **Association of European Anæsthetists** held its second Congress in Lyon on 12 July 1959.

Communications were given by: J. Bonica (U.S.A.): 'The public relations of the anæsthesiologist'. G. Organe: 'The organisation of the anæsthetic services in Great Britain.' Dr Martin: 'The instruction programme for the anæsthesiologist in the U.S.A.'

There was a discussion on the place of the anæsthetist in modern medicine.

The **Mexican Society of Anesthesiologists** is holding its eighth congress jointly with the fifth Latin-American congress at the University City in Mexico DF, in November 1960. Fellows and Members of the Association of Anæsthetists of Great Britain & Ireland are invited to attend or to submit scientific articles. All correspondence should be addressed to Al Apartado Postal 13649, Admon, 45, Mexico 1, DF.

Published by
THE ASSOCIATION OF ANÆSTHETISTS OF GREAT BRITAIN & IRELAND
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Published bi-monthly, one volume a year, by
THE WILLIAMS & WILKINS COMPANY
Baltimore, Maryland, U.S.A
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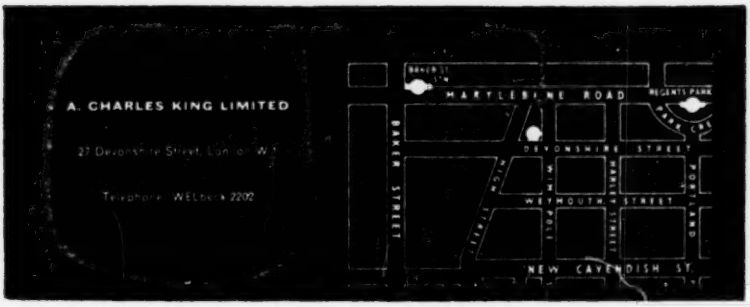

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24, Thayer Street, London, W.1.